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The Journal OF Nervous and Mental Disease

An American Journal of Neuropsychiatry, Founded in 1874

ORIGINAL ARTICLES

EXPERIMENTAL STUDIES INDICATING AN INFECTIOUS ETIOLOGY OF SPASMODIC TORTICOLLIS

BY EDWARD C. ROSENOW

DIVISION OF EXPERIMENTAL BACTERIOLOGY, THE MAYO FOUNDATION,
ROCHESTER, MINNESOTA

During the course of numerous experiments with the streptococcus from encephalitis, epidemic hiccup, and chorea, a small percentage of animals developed peculiar movements of the head. These movements usually developed some time after the acute symptoms of encephalitis had subsided, as is sometimes the case in man. Believing that spasmodic torticollis might be due to microorganisms which have specific localizing power, I decided to study the etiology of this disease from the bacteriologic standpoint. The methods used were similar to those employed in my studies in encephalitis and epidemic hiccup.(7) Suffice to say, that intracerebral or subdural inoculations of suspensions in sodium chlorid solution of nasopharyngeal swabs, of pus expressed from tonsils, or aspirated from the depths of pyorrheal pockets, and cultures from these were chiefly employed, the method which has yielded positive results in the case of virus studies, especially in poliomyelitis. Intravenous injections of the respective cultures were made in a small number of animals, usually after facilitating penetration of the meninges by subdural injections of sterile water or normal horse serum. Filtrates of washings from the nasopharynx of patients, and of the brains of rabbits that developed typical symptoms, and highly diluted cultures after many rapidly made subcultures were also injected intracerebrally.

ILLUSTRATIVE CASES

Case 1. A man, aged thirty-three years, came to the Clinic April 25, 1921, because of an uncontrollable impulse at frequent intervals to turn his head downward and to the right. The trouble had begun six weeks before, shortly after an attack of stiff neck, which had lasted for several days. The patient complained of difficulty in keeping his head directed forward, especially when excited, or when riding. At no time were these attacks associated with pain. About ten days before my examination, he had been nearly free from the symptoms for about half an hour, and for short intervals since. He had had ten chiropractic treatments, and injections of atropin, without relief, and the condition steadily progressed. His general health was good, his appetite normal, and he was not subject to headache. The attacks did not occur at night, and he slept well.

Examination: Quite regularly, while talking, the patient's head



FIG. 1. Marked rotation of head and eyes in rabbit 2616 six months after intracerebral injection.

was drawn slowly to the right and downward, and brought back into normal position more quickly. When asked why this was done, he said that it was impossible for him to control the impulse, and that after his head had turned, the impulse was relieved temporarily. The pupils were equal and reacted normally to light and accommodation. His pulse was 100, temperature 99.2° on one occasion, and 98.6° on another. The systolic blood pressure was 120, the diastolic 90, the hemoglobin 75 per cent, the leukocyte count 10,200. The Wassermann test was negative, and the urine normal. The knee jerks were slightly exaggerated, but otherwise the examination was negative. Two teeth were slightly tender. His throat was moderately red; the tonsils were enlarged, and from the left, fluid pus was expressed. Diagnosis of spasmodic torticollis was made. A vaccine was prepared from the strain which was isolated from the pus from the tonsils, and which produced in animals a condition similar to that of the patient and tonsillectomy was advised.

Experiments: The salt solution suspension of the pus expressed from the tonsils was injected directly intracerebrally into two rabbits, and the primary glucose-brain-broth cultures intraperitoneally into two mice. The mice remained well. Both rabbits developed peculiar lateral movements of the head, marked tremor, ataxia, and, shortly before death, increased respirations and weakness of extremities. Necropsy revealed congestion of the vessels of the meninges, and hemorrhagic edema of the lungs. Cultures from the blood were negative; those from the brain yielded countless numbers of green-producing streptococci and a few staphylococci.

One-tenth cubic centimeter of a 1:50 to 1:5000 dilution in sodium chlorid solution of a 5 per cent emulsion of the brain of one of these

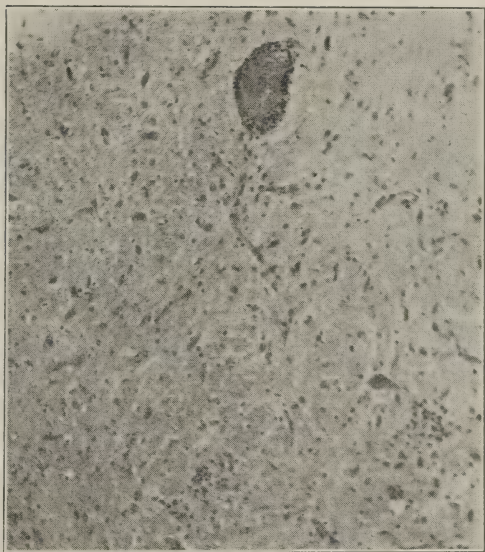


FIG. 2. Localized and perivascular infiltration in the medulla of rabbit 2616.

rabbits was inoculated intracerebrally into four rabbits. All developed marked movements of the head, and other symptoms similar to those described in the protocol of rabbit 2615.

Rabbit 2615 was injected April 30, 1921, with 0.1 c.c. of a 1:50 dilution of the emulsion of the brain of the rabbit that developed torticollis following injection of the pus from the patient's tonsils. May 1, at 8 A.M., the animal was found lying in the cage, with marked weakness of the hind extremities. It almost continually slowly moved its head laterally in a somewhat jerky fashion, more often to the right, and synchronously with these movements the muscles of the ears contracted sharply, bringing the ears erect, followed by relaxation, each time the head was brought into normal position. During some of the movements of the head, there was sharp contraction of the muscles of the shoulders and, synchronously,

of the lower jaw, causing the animal to open and close its mouth. At 12 M. the condition was unchanged, except that the fore extremities were definitely weak. At 4 P.M. the lateral movements of the head continued, and all extremities were extremely weak. The next day the animal was found dead. The spinal fluid was distinctly turbid. A moderate number of small hemorrhages were found in the cervical cord and in the medulla, and a large number in the lumbar enlargement. Cultures of the pipetted material from the brain and the spinal fluid yielded numerous colonies of the green-producing streptococcus in pure culture; those from the blood were sterile. An ascites-tissue-fluid culture of a filtrate of the emulsion of the brain produced a cloud in the lower half of the tube at the end of five days, due to an extremely pleomorphic streptococcus. Sections revealed leukocytic and round-cell infiltration and gram-positive diplococci in the brain (Fig. 15a).

This filtrate, which yielded a pure culture of the streptococcus, was injected into two rabbits. One remained well; the other died



FIG. 3. Peculiar positions of the head of rabbit 3070; *a*, three, *b*, fourteen, and *c*, forty-nine days after intracerebral injection, in sharp contrast to that of normal control.

within twenty-four hours. Necropsy disclosed a large cerebral hemorrhage surrounding the place of inoculation, and widely disseminated hemorrhagic edema of the lungs. Cultures from the brain yielded the streptococcus. The highly diluted emulsion of the brain of rabbit 2615 was injected intracerebrally into three rabbits. Two died in forty-eight hours from meningo-encephalitis without developing torticollis. The protocol of the third rabbit follows:

Rabbit 2616 was injected May 1, 1921, with 0.1 c.c. of a 1:1000 dilution of the emulsion of the brain of rabbit 2615 (third animal passage). May 2, at 7 A.M., the animal appeared well. At 8 P.M. it sat quietly in the cage, apparently afraid to hop, and was slightly tremulous when made to do so. Respirations were increased and at repeated intervals peculiar jerky movements of the fore part of the body, and, at longer intervals, drawing of the head to the right were noted. At 9 P.M. mild clonic spasms of the muscles of the abdominal wall and back had developed, which the animal apparently attempted to control by assuming different positions. There was an occasional blepharous spasm of the right eye synchronous with spasms of the muscles of the right side of the neck. May 3, at 2 A.M., the condition

was about the same. At 6 A.M. it was undoubtedly better; the spasms had largely disappeared, and the movements of the head were less marked. At 12 M. the head was tilted slightly to the left, and the peculiar movements continued. At 4 P.M. the turning and tilting of the head were unchanged, but contractions of the muscles of the fore extremities had developed, which at times almost threw the animal over, and which it attempted to control by crouching to the floor of the cage and extending its extremities. At 8 P.M. the animal was quiet, but when prodded the symptoms returned. May 4, at 7 A.M., the general condition was undoubtedly better. The animal sat quietly in a crouched position, but appeared afraid to move, and when induced to do so peculiar violent contractions of the muscles of the fore extremities and marked drawing of the head to the left developed, and became worse when the animal was placed on a smooth surface. When returned to its cage, it crouched to the floor, apparently in an effort to control the muscular contractions and movements



FIG. 4. Peculiar positions of the head of rabbit 3134; *a*, relatively normal, *b*, moderately extended, and *c*, markedly retracted and twisted, with fore part of body elevated, in sharp contrast to normal control.

of the head. On repeated observations, it was found relatively quiet in its cage, but if prodded the symptoms and abnormal muscular contractions recurred. May 5, the animal was extremely unsteady; it tended to turn to the right, and on prodding, extreme ataxia developed, as a result of sharp clonic contractions of the muscles of the fore extremities. May 6, the animal appeared well. It ate oats and carrot with relish. The ataxia had become less severe, but the lateral movements of the head were more marked. At intervals, when the animal sat in a crouched position, apparently asleep, the movements of the head were entirely absent, but when prodded, or when aroused by placing a carrot to its mouth, marked lateral and up and down movements of the head became manifest, making eating impossible. On repeated occasions, stroking the animal slowly seemed to enable it to control the movements of the head. May 7 the condition was unchanged. From May 7 to 18 a noticeable change in the animal's condition developed. The spasmodic contractions of the muscles of the fore extremities disappeared entirely, but the peculiar tic-like movements of the head continued and were unduly affected by surroundings. During this time, therefore, the effect of external

stimuli carried to the centers through the senses of sight, hearing, and smell was frequently tested. It was found that when observed unnoticed the animal remained quiet over long periods, except for short intervals of relatively slight spasmodic movements of the head, which disappeared during sleep. My entrance into the room, without going near the cage, the smell of carrot, of which it was very fond, and external stimuli, such as teasing by pulling its "whiskers," or placing food before its mouth and then taking it away, were often followed by extreme movements of the head, which grew progressively worse, and not infrequently caused the animal to throw itself completely over. These tantrums would cease abruptly when the animal was petted by stroking its fur, or by allowing it to eat carrot. May 25 the condition was about the same, but the head and eyes were rotated toward the right. The head could be brought into normal position by overcoming considerable resistance, but when released the

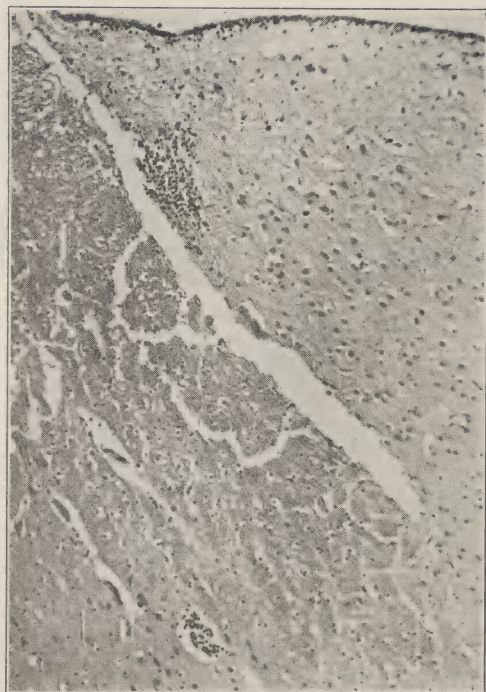


FIG. 5. Localized and perivascular infiltration adjacent to the lateral ventricle in rabbit shown in Figure 4.

abnormal position was resumed. From this date until November the torsion of the head continued (Fig. 1). The spasmodic movements of the head became progressively less marked. The animal died apparently of intercurrent cause, November 9. The brain and cord were normal, except for a large brownish area in the pia over the

anterior and right aspect of the medulla. Cultures from the brain and blood were sterile. Marked localized perivascular and parenchymatous round-cell infiltration were found in the subcortical region of the cerebrum on the right side opposite the lower rolandic area, and unilateral perivascular and localized parenchymatous infiltration in the medulla (Fig. 2) and at the juncture of the cerebellum and pons.

The total incidence of lesions in the nine rabbits injected with material directly from the tonsils and after one and two animal passages is summarized in the table.

Comment: Points of special interest in this case are the course of the patient's attack, associated with what is usually regarded as acute wry neck, the absence of pain, and the high incidence of the development of the tic-like movements of the head in the animals injected.

Case 2. A woman, aged forty-three years, came to the Clinic October 14, 1921, on account of convulsive spasms of the neck and mouth, and soreness and pain in the back of the neck. The patient had been well until 1917, when she suffered from an anal fissure following childbirth. She was then somewhat run down, and began to notice intermittent drawing sensations in the right corner of her mouth. These twitchings, which were slight and not noticeable to others, but easily felt by the patient, would continue for a few days at intervals of several weeks. During the summer of 1918 she was almost entirely free from them, but during the winter of 1918 to 1919, when under great mental stress, the attacks occurred more frequently, and within a year extended to the muscles of the back of the neck, which became sore and painful. Since June, 1921, the trouble had progressed markedly and had spread down the back to the angle of the scapula on the right side. The spasms ceased entirely during sleep, and were better in the morning, but were always worse when the patient became tired from physical or mental exertion.

Examination: The patient was found to be well nourished, and appeared well, but at intervals had tic-like movements of the right side of the face and back of the neck, and her head was pulled rather sharply backward and to the left. In these attacks, the right sternomastoid and the muscles of the back became very tense. A loose right kidney, lacerated cervix, internal hemorrhoids, and tenderness in the region of the appendix were noted. The heart and lungs were normal, and the knee jerks active. The temperature was normal, pulse 72, hemoglobin 71 per cent, erythrocytes 4,280,000, leukocytes 7,600, differential count of leukocytes normal, and the urine normal. Roentgenograms of the sinuses of the head, chest, and pelvis were negative, but indications of slight hypertrophic arthritis of the cervical spine were found. Many of the teeth had been filled, but otherwise were normal. The tonsils were small and ragged, and from both liquid pus was expressed. Tonsillectomy was performed, animals

were inoculated, and a vaccine was prepared from the organism with which torticollis was produced in the animals.

Experiments: Sodium chlorid solution suspensions of the nasopharyngeal swab, of the pus expressed from the tonsils, and of the emulsion of the tonsils, were injected intracerebrally into four rabbits. Three of these developed tic-like movements of the head; one remained free from symptoms. The one injected with material from the nasopharynx died in three days. Necropsy revealed marked edema and infiltration of the pia over the upper end of the medulla at the juncture with the cerebellum. Lesions of the muscles or the nerves of the neck were not found. Cultures from the brain yielded countless numbers, those from the blood a few colonies of the green-producing streptococcus. The primary culture in glucose-brain-broth of the blood of this rabbit was injected into two rabbits. Both developed abnormal movements of the head, illustrated in the protocol of rabbit 3070.

Rabbit 3070, weighing 1,160 gm., was injected October 21, 1921, with 0.2 c.c. of a 1:1000 dilution of the glucose-brain-broth culture of the blood. October 22 the animal appeared well during the day, but at 9 P.M. was somewhat unsteady, and moved its head slightly to the left at repeated intervals. October 23, at 8:30 A.M., it sat quietly, holding its head rather high and in a retracted position. At 12 M. the head was still held high, rhythmic movements backward and to the left had developed, and each time as the head was brought backward the body was raised slowly to the left, often almost causing the animal to fall, when it would catch itself and bring the head sharply forward and immediately again into a retracted position. There was moderate hyperesthesia, slight stimulation causing marked increase in the movements of the head, the animal tending to lose its balance. At 9:30 P.M. the condition was practically unchanged, but fine tremor, more marked on the left side, and occasional sharp contractions of the muscles of the neck were noted. October 24, at 7:30 A.M., it was found sitting quietly, with the head in a retracted position and the body leaning to the left. It brought its head forward to eat, and then back (Fig. 3a) as it chewed its food with apparent relish. The peculiar movements of the head, relatively slight when quiet, completely absent in sleep, and usually worse on prodding, were noted on repeated daily observations. November 3 a bald spot appeared on the back of its neck. From November 3 to 29, when the animal was chloroformed, a notable change in the movements of the head developed. It appeared to have greater difficulty in bringing the head backward (Fig. 3b), and a gradual tendency to bring it abnormally forward and downward developed. Frequently the head was brought far under the body (Fig. 3c), and the animal could scarcely bring it into normal or retracted position. No lesions were found at necropsy. Cultures from the blood and the brain remained sterile, and microscopic examination revealed only slight localized mononuclear infiltration among the ganglion cells on the lateral aspect of the cerebral cortex, and slight perivascular

round-cell infiltration and localized infiltration along the floor of the fourth ventricle. The results after three animal passages, and many subcultures are illustrated in the protocol of rabbit 3134.

Rabbit 3134, weighing 1,210 gm., was injected intracerebrally November 5, 1921, with 0.2 c.c. of a 1:100 dilution of the 5 per cent emulsion of the brain of a rabbit inoculated with the patient's strain in the second animal passage and in the forty-fourth subculture (third animal passage). November 6, 7, and 8, the animal appeared well. November 9 it seemed somewhat slow but otherwise well. November 11 there was slight tilting of the head to the right and a tendency to hold it rather high and in retracted position. The animal appeared distinctly slow and was disinclined to move, and when made to do so, increased tonus of the muscles of the extremities and slight ataxia were noted. November 12 it sat quietly as if half asleep and responded slowly to stimuli. It was distinctly ataxic, the fore part of the body tended to bob, and the tonus of the muscles was increased.



FIG. 6. *b* and *c*, peculiar positions of the head of rabbit 3172; and *a*, normal control injected with the filtrate.

Prodding induced peculiar movements of the head, in which it was tilted slightly to the left and held high, with the left ear drooping. There was congestion of the conjunctiva and moderate lacrimation. November 14 the animal appeared less lethargic and responded more quickly to stimuli. It drank and ate normally, but the movements of the head continued intermittently. November 15 the condition was about the same. The animal sat quietly for a time with the head in normal position (Fig. 4*a*), but repeatedly, without apparent cause, the head was lifted and retracted (Fig. 4*b*), thrown forward and to the side several times and then turned to the left and brought backward as the forepart of the body was raised, often almost to the point of causing the animal to fall over its haunches (Fig. 4*c*). Following this extreme position, it sat quietly for a short interval, when this cycle was repeated. Excitement or prodding induced the attacks on repeated occasions. The condition continued unmodified until November 22, when the animal was found dead. Necropsy revealed

no changes in the brain, cord, cervical nerve trunks, nor muscles of the neck. Cultures from the brain and blood were negative.

Sections revealed localized areas of round-cell infiltration in the deep layers of the cerebrum on the inner aspect of the wall of the left lateral ventricle (Fig. 5), and in the cerebellum near the juncture with the pons, slight infiltration by round cells between the ganglion cells in the cerebral cortex, and a few diplococci in the lesions (Fig. 15*b*).

Of the twenty-two rabbits injected intracerebrally with this strain directly from the throat and tonsils, with the emulsion of the brain, or with cultures from positive rabbits, including many subcultures, twelve (55 per cent) developed peculiar tic-like movements of the head, eleven ataxia, eight tremor of muscles, eight marked weakness, six turning of the head, and four lethargy (Table). Torticollis



FIG. 7. Peculiar posture, and position of the head of rabbit 3264, four days after intracerebral injection of the streptococcus from Case 4.

developed during three successive animal passages, and as high as the forty-fourth subculture. Only one of the four rabbits that developed lethargy was injected with the strain as isolated, two when in the second passage and the forty-fourth subculture, and one with this strain after three animal passages.

The filtrates of the emulsion of the tonsil and of the brain of one positive rabbit proved sterile and were injected intracerebrally into two rabbits each. All remained well.

Three rabbits were injected intravenously with the strain from this patient. One received 2 c.c. of the glucose-brain-broth culture of the blood of a positive rabbit injected with a suspension of the pus of the patient's tonsils. The animal developed marked tremor and twitching of the muscles of the fore extremities and neck, and difficult, gasping respirations. No gross lesions of the brain were found at necropsy. Cultures yielded the characteristic streptococcus.

SUMMARY OF RESULTS FOLLOWING INTRACEREBRAL INOCULATION OF MATERIAL CONTAINING THE STREPTOCOCCUS FROM SPASMODIC TORTICOLLIS

Case	Rabbits		Mortality per cent	Spasms of muscles			Abnormal movements and posture						Tremor	Restlessness	Hyperpnea	Convulsions	Paralysis	Lethargy	Animals cultured	Characteristic streptococcus	
	Injected	Died		Diaphragm	Abdominal	Other	Nystagmus	Rhythmic movement of head	Tic-like movement of head	Ataxia	Turning of head	Retraction of head									
1	9	7		0	2	3	0	1	7	6	2	0	4	2	2	0	3	0	9	8	4
2	22	12		0	1	3	1	1	12	11	6	3	8	1	3	0	8	4	17	8	2
3	19	12		0	2	2	4	4	10	9	7	2	6	1	6	0	3	0	13	8	4
4	8	5		0	0	1	2	1	5	5	2	3	2	0	4	0	3	0	8	6	3
5	4	4		0	0	1	1	2	3	3	2	2	3	0	3	0	2	0	3	2	0
6	4	4		0	0	0	0	1	3	4	0	2	3	0	2	0	1	0	4	4	0
7	2	2		0	0	0	0	0	1	2	0	0	1	0	0	0	1	0	2	1	0
8	2	2		0	0	0	0	0	1	1	1	0	1	0	1	0	1	0	2	2	0
TOTAL	70	48	69	0	5	10	8	10	42	41	20	12	28	4	21	0	22	4	58	39	13
Normal controls (77)	106	24	23	0	0	2	7	1	3	17	19	2	4	2	10	0	8	0	97	24	7

The other two rabbits were given 3 c.c. of the same strain after sixteen rapidly made subcultures, one also receiving 0.5 c.c. of normal horse serum intracerebrally. The one receiving the culture only remained well. The other died the day after injection without developing the tic-like movements of the head. The streptococcus was isolated in countless numbers from the brain; the blood was sterile.

Comment: Seven months after tonsillectomy the patient returned for observation, having taken increasing doses of the vaccine meanwhile. There was undoubted improvement. The muscles of the neck were more relaxed; the contractions were less severe and less frequent and the mucous membrane of the nasopharynx was normal.



FIG. 8. Extreme retraction of the head in rabbit 3300 injected four days previously with the streptococcus from the rabbit shown in Figure 7.

A salt solution suspension of two swabs from the nasopharynx taken two days apart were each injected intracerebrally into two rabbits without effect.

Case 3. A man, aged forty-four years, came to the Clinic November 7, 1921, on account of severe pain in the back of the neck, and backward jerkings of the head, which had begun gradually nearly three years before. He had taken various medicines and treatments without relief. After nine months he had had to stop work. One year after the onset, an incision was made over the left clavicle with apparently no definite purpose and without relief. His tonsils were removed, and the following summer he went back to work, but the pain continued, and work was difficult because his head was twisted to one side. July, 1921, the tendons were cut on both sides above the clavicle, and the sternomastoid on the left side was removed. Since that time his head had not twisted so far to the right, but the pain

and twitchings continued. He thought that his head twitched about the same whether he moved or was quiet, but when his mind was intensely occupied, as in reading an interesting book, the twitchings became less for a time; and they disappeared during deep sleep. The attacks recurred at regular intervals of from two to five minutes; first a slight contraction of the muscles of the face, then of the neck, which grew rapidly worse, until the face was markedly distorted and the head turned rather sharply upward and to the left as the shoulders were moved from side to side. This was followed by almost complete relaxation for a short interval.

General examination was negative except that the pulse was rapid and a roentgenogram revealed two impacted teeth; these were removed by surgical resection.

Experiments: Suspensions in sodium chlorid solution of swabs from the tonsil and the nasopharynx were injected intracerebrally into four rabbits. Peculiar movements of the head developed in three, two of which died in forty-eight hours, and one in nine days. Necropsy revealed leukocytic infiltration, especially in the pons and medulla, in the first two, and round-cell perivascular infiltration in the other. The fourth, injected with 0.1 c.c. of a 1:1000 dilution, remained well, and no microscopic changes were found in the brain. Of seven rabbits injected with the culture from the outside of one impacted tooth, not one developed abnormal movements of the head, but all died within forty-eight hours, of meningitis. Eight rabbits were injected with the culture of the streptococcus from the pulp of the impacted tooth in the first and second animal passages, two of which included the strain in the third subculture. Of these, seven developed peculiar movements of the head; illustrative protocols follow:

Rabbit 3160, weighing 1,530 gm., was injected intracerebrally November 25, 1921, with 0.2 c.c. of a 1:1000 dilution of a glucose-brain-broth culture of the pulp of one of the impacted teeth. November 26 the animal appeared well. November 27, at 9 A.M., it was disinclined to move, seemed uncomfortable, and crouched repeatedly, apparently in an attempt to find a comfortable position. Respirations were slightly increased. At 11 A.M. rhythmic vertical nystagmus had developed, synchronous with lateral movements of the head. At 7 P.M. the nystagmus was more marked, especially in the left eye, and synchronously the head rotated to the right. At 9 P.M. vertical nystagmus of the left eye was still marked, the right eye was completely closed, and the head rotated to the right, but could be brought to its normal position by overcoming marked resistance. When prodded, the animal tended to roll over and over, always to the right. November 28, at 12 M., the nystagmus had disappeared, but the rotation of the head continued. The animal repeatedly drew its left leg sharply up toward the side of the face. At 4 P.M. there were repeated vertical oscillations of both eyeballs, and the animal seemed to have difficulty in swallowing. November 29 it was found dead. Necropsy disclosed marked congestion of the vessels of the meninges, moderate

edema and infiltration of the pia over the anterior aspect of the medulla, and numerous small punctate hemorrhages in the lower portion of the medulla and upper cervical cord. Cultures from the brain yielded the characteristic streptococcus; those from the blood were sterile. Sections from the brain revealed moderate leukocytic and round-cell infiltration, most marked in the medulla and pons, and diplococci in the lesions; those of the middle and internal ear showed no changes.

Rabbit 3172, weighing 1,080 gm., was injected intracerebrally November 30, 1921, with 0.2 c.c. of a 1:1000 dilution of the glucose-

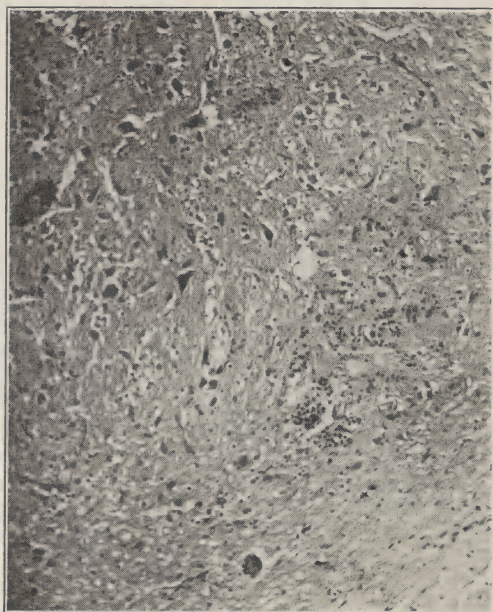


FIG. 9. Leukocytic and perivascular round-cell infiltration in the subcortical region of rabbit shown in Figure 8.

brain-broth culture of the brain of rabbit 3160 after three rapidly made subcultures. December 1, at 9 A.M., the animal sat quietly, with head tilted somewhat to the right, and rhythmic horizontal nystagmus of both eyes, synchronous with drawing of the head to the left. When prodded, it was excitable and somewhat ataxic, and the lateral movements and tilting of the head became very much worse. At 11 A.M. the nystagmus was less marked. The head was lowered slowly and rhythmically and pulled rapidly upward and to the right, even at times so far backward that the animal fell over its haunches (Fig. 6 *b, c*). These attacks recurred frequently and varied greatly in intensity. Especially after goading, the symptoms became more severe up to a certain point, when almost complete relaxation followed for a short interval. At 8 P.M. the condition was

about the same. The peculiar movements of the head were more regular, each cycle taking approximately fifteen seconds. The animal ate oats, and while it chewed, its head was thrown backward and to the right once or twice before it was brought forward to take more food. December 2, at 8 A.M., the nystagmus had entirely disappeared, but the movements of the head continued. At 2 P.M. the animal was weaker. At 4 P.M. the movements of the head had ceased, but marked weakness and repeated clonic spasms of the abdominal muscles had developed. At 4:30 P.M. the animal was found dead. Necropsy revealed marked congestion of the vessels of the meninges, edema and infiltration of the pia on the posterior aspect of the cerebellum and medulla and over the upper cervical cord, and a few small hemorrhages in the cervical cord. Cultures of the blood and brain yielded the organism injected.

Comment: Of the nineteen rabbits injected intracerebrally with this strain in the first and second animal passages, including the seven negative rabbits injected with the culture from the outside of the impacted tooth, ten developed peculiar tic-like movements of the head; nine, ataxia; seven, turning of the head; six, hyperpnea; six, tremors; three, twitching of muscles; and three, untoward weakness (Table). Four rabbits were injected in the anterior chamber of the eye after intracerebral injection of sterile horse serum. Penetration of the meninges did not occur in any. Seven were repeatedly injected intravenously with 0.01 to 0.1 c.c. doses of glucose-brain-broth culture without effect. Four were injected intravenously with one large dose (1 to 5 c.c.) of the broth culture. Two of these were given also sterile horse serum intracerebrally. The two receiving the culture only remained well; the two receiving the horse serum developed an almost identical train of symptoms, and the necropsy and cultural findings were identical. The animals became restless, tremulous, and hyperesthetic; the head was drawn repeatedly to the right, and respirations increased markedly. The pia over the posterior and lower part of the cerebellum, medulla, and cervical cord was edematous and cloudy. Cultures from the brain and spinal fluid yielded countless numbers of the streptococcus; the blood was sterile.

Case 4. A man, aged thirty-eight years, came to the Clinic January 10, 1922, because he became easily fatigued. His expression was abnormal, especially on the right side, when he laughed or sang, and his head twitched. The symptoms had begun about one month before, with chills, malaise, fever, hoarseness, loss of appetite, and general debility, eight months after apparent recovery from an attack of encephalitis. Following this for about six weeks he had diplopia and blurred vision. At the onset he was restless and irritable and

for a time was unable to sleep, but this was followed by a period of lethargy, when he slept almost continually for two and a half weeks. During this time he would wake each night on account of difficult breathing, rapid, labored respirations, and sudden jerking of his legs. After three weeks in bed, he slept less and began to improve. Six weeks later he was again able to walk and thought himself well.

Examination: The general examination was negative except for

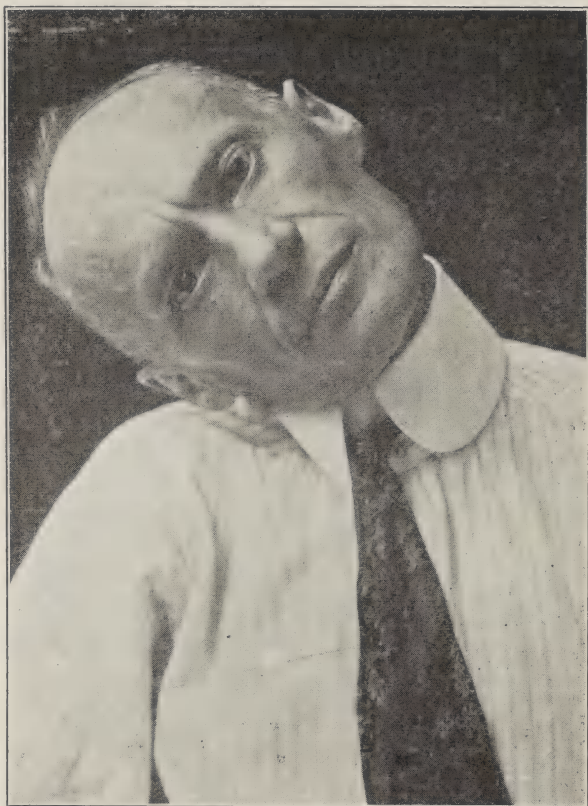


FIG. 10. Marked tilting and slight retraction of the head (Case 5).

evidence of involvement of the left third and sixth and the right seventh nerves, an Argyll-Robertson pupil, and buried tonsils, from each of which pus was expressed. His memory for names had become slightly defective. His head was held slightly to the right and at times oscillated from right to left. In laughing, both sides of his face became distorted, the right eye closed more than the left, and that side of his face wrinkled more than the other, giving, as he described it, an appearance of pain. The right palpebral fissure was slightly narrower than the left. The eyegrounds were normal; the hemoglobin 74 per cent; erythrocytes 4,460,000; leukocytes 8,800;

blood Wassermann test negative; and the urine normal. The patient was recovering from an attack of acute follicular tonsillitis.

The pus expressed from the tonsils yielded numerous colonies of hemolytic streptococci and only a few of green-producing streptococci. The latter, in the third subculture in glucose-brain-broth, was injected intracerebrally into two rabbits. One, receiving 0.2 c.c. of the undiluted culture, died during the night, of meningitis; the other (rabbit 3264) developed symptoms.

Experiments: Rabbit 3264, weighing 1,070 gm., was injected January 26, 1922, with 0.1 c.c. of a 1:100 dilution of the glucose-brain-broth culture. January 27, at 8 A.M., the animal appeared well but repeatedly moved its head backward and to the right. When prodded and forced to hop, the movements of the head became extreme, and the animal maintained its balance with difficulty. When quiet after these attacks, rhythmic, relatively slight movements of the head occurred. At 2 P.M. the condition was practically unchanged, except that the head had become unsteady. At 8 P.M. the animal sat quietly with the head slightly retracted; it repeatedly twisted its head, bringing it upward and to the right in retraction and then suddenly forward, repeating this several times in succession before bringing it to normal position. When prodded, it became excited and the movements more marked. January 30 respirations were normal. It sat quietly, hopped and ate readily, but when prodded, and occasionally at other times, it threw its head backward and to the left. Occasionally it became excited and ran around in a circle, always to the left, shaking its head. Each time before becoming quiet, it threw its head violently backward in rapid succession a number of times. January 31 it held its head in retracted position most of the time, and constantly tended to stand with the fore extremities on its mate or some other object (Fig. 7). The condition remained unchanged until February 7, when the animal was found dead from intercurrent colon bacillus infection. Necropsy did not reveal lesions of the brain, cord, nerves, or muscles of the neck, but liquid contents were found in the cecum, which was markedly distended. Cultures of the blood and brain contained a large number of colon bacilli.

The streptococcus in the third subculture from the blood of the mate to rabbit 3264 was injected into six rabbits. Two of these died within twenty-four hours from meningitis, whereas the other four lived three, four, six, and thirteen days and developed marked tic-like movements of the head. Sections revealed perivascular round-cell infiltration. The protocol of rabbit 3300 suffices to illustrate.

Rabbit 3300, weighing 1,580 gm., was injected February 2, 1922, with 0.1 c.c. of a 1:1000 dilution of the third rapidly made glucose-brain-broth culture after one animal passage. February 3, the animal sat quietly, apparently well, but when made to hop, it crouched repeatedly, pushing its head into a corner of the cage, in which position it would remain for long periods. February 4 the condition was about the same. February 5 it no longer tended to brace its head and, instead, held it in a retracted position most of the time. February 6,

at 8 A.M., it was quiet and usually held its head in a sharply retracted position. When prodded, it threw its head backward and forward many times, and when it hopped the movements of the head became more marked, and ataxia developed. At 5:10 P.M. the head was turned sharply backward between its shoulders (Fig. 8), but was readily brought forward when the animal ate. When the head was in the retracted position, violent grinding of the teeth was noted. The tension of the muscles that drew the head backward varied greatly, as evidenced by the alternately slight and great resistance noted on grasping the head and bringing it forward. When the



FIG. 11. Sharply demarcated hemorrhage, edema and infiltration of the pia over the anterior aspect of the medulla and pons, *a*, in a rabbit injected subdurally far forward over the right frontal lobe with the sodium chlorid solution suspension of the swab from the nasopharynx (Case 5).

animal became excited, it had difficulty in separating its jaws in attempting to eat, and repeated retraction of the head developed, even to the point of causing the animal to fall backward. February 7 it was observed most of the day. The retraction of the head was more marked. It was brought back slowly as the fore part of the body was elevated and the fore extremities held in extension. This was frequently associated with lateral movements of the head and clonic spasms of the muscles of the face, more marked on the right side, while the animal threw itself to the side as it developed clonic spasms, beginning with the muscles of the right extremities. Later in the day, after sitting quietly for a time, the clonic spasms and retraction of the head became more marked, and repeated epileptiform seizures developed, in which respiration ceased, the extremities stretched in a tonic manner, and the head flexed. On recovery from these attacks

respiration was resumed, and clonic twitchings of the muscles of both sides of the face, the eyelids, and the fore extremities developed. February 8 the animal was found dead. Necropsy revealed marked congestion of the vessels of the meninges, edema and infiltration on the anterior aspect of the medulla, pons, and posterior portion of the cerebellum, and a few minute hemorrhages on the anterior part of the medulla and the upper cervical cord. Cultures of the blood were sterile; those from the brain yielded a pure growth of the streptococcus but produced granular growth in glucose-brain-broth instead of diffuse turbidity. Sections revealed widespread perivascular and localized parenchymatous infiltration by leukocytes and round cells, most marked in the subcortical region (Fig. 9) of the cerebrum, and in the medulla, and a moderate number of gram-positive diplococci in the lesions (Fig. 15d).

The cultures from the emulsion of the tonsil yielded chiefly hemolytic streptococci, and the patient developed severe pharyngitis following tonsillectomy. The three rabbits injected with the emulsion died of suppurative meningitis induced by hemolytic streptococci. The filtrate of the emulsion of the tonsil proved sterile and was without effect in two rabbits injected. They were chloroformed after nineteen days, and sections from the brain were negative.

Of the eight rabbits injected with the green-producing streptococcus from the tonsil pus in the third subculture, in the first and second animal passages, five developed peculiar movements of the head; five, ataxia; four, hyperpnea; two, tremor of muscles; two, nystagmus; two, turning of the head, and three, marked weakness (Table).

Comment: The points of particular interest in this case are the isolation of the characteristic streptococcus in spite of the presence of acute hemolytic streptococcic pharyngitis following tonsillectomy; death from meningitis in the rabbits injected with the hemolytic streptococcus, and the negative results following the injection of the filtrate of the emulsion of the tonsils.

Case 5. A man, aged fifty-seven years, came to the Clinic February 22, 1923, on account of difficulty in swallowing and peculiar movements of the head. His trouble began six months before with a feeling of pressure and pain in the occipital region; two months later he developed an uncontrollable tendency to pull his head from side to side continuously (about sixty times each minute). This condition, which was associated with extreme restlessness, and at times shortness of breath, persisted for several months and then disappeared. Six weeks prior to coming to the Clinic, he developed difficulty in swallowing, chewing, and speaking. On drinking water, it frequently regurgitated through his nostrils. His voice had changed, and he developed abnormal movements of the head, in which it was repeatedly drawn to the right and backward (Fig. 10).

Examination: The patient was hyperesthetic and complained of

occipital pain. At the time of examination his voice was low and hoarse; the muscles of the pharynx were relaxed; swallowing was difficult; the tonsils were small and covered by the anterior pillar; there was moderate pyorrhea; the tongue showed tremor and slight atrophy and deviated to the right; the pupils were equal and reacted normally to light and accommodation. The leukocyte count was 5,500; the temperature was normal; the urine contained a moderate amount of sugar, and the blood Wassermann reaction was negative.

Experiments: Two rabbits were injected with the sodium chlorid solution suspension of the nasopharyngeal swab, one receiving 0.2 c.c., the other 0.1 c.c., and two with similar suspension and dosage of material aspirated from the depths of pyorrheal pockets. The symptoms were similar in all. The two receiving the larger dose



FIG. 12. Marked retraction and slight tilting of the head to the left in rabbit 4366, two weeks after subdural inoculation with the sodium chlorid solution washings of the swab from the nasopharynx (Case 5).

died in three days, the other two in eighteen and twenty-nine days, respectively. All developed marked disturbance of respiration and peculiar movements of the head, three paralysis, beginning in the fore extremities, and two, tic-like movements of the head as a late manifestation. In the first two, the pia on the anterior aspect of the medulla and pons was infiltrated (Fig. 11), and cultures yielded a pure growth of the streptococcus. Necropsy findings of the other two animals were negative, and the cultures remained sterile. The protocol of rabbit 4366 will suffice to illustrate.

Rabbit 4366, weighing 1,650 gm., was injected subdurally February 28, 1923, with 0.1 c.c. of the salt solution suspension of the nasopharyngeal washings. March 1 the animal was somewhat tremulous, and respirations were increased. March 2, at 8 A.M., it pulled its head to the left frequently and shook it when hopping, at

the same time manifesting undoubted weakness of the fore extremities and irregular increased respirations. At 8 P.M. the condition was about the same. Movements of the head backward and to the left continued at irregular intervals and became worse on prodding. March 3, at 7 A.M., slight tic-like movements of the head were noted, and the animal was somewhat tremulous on hopping. March 14 it had apparently recovered completely, except for the peculiar movements of the head. It sat quietly, holding its head backward most of the time (Fig. 12). After prodding or teasing, such as taking its food away from it, it developed tantrums in which the motions of the head became very severe, but quieted when its fur was stroked. The condition remained the same until March 28, when it was found dead, apparently from intercurrent cause. There was marked distention of the large intestine and the stomach, associated with numerous hemorrhages. There were no microscopic lesions other than perivascular infiltration surrounding the fourth ventricle high up in the medulla, and slight perivascular infiltration of the pia over the anterior aspect of the medulla, pons and cerebellum.

Comment: The points of particular interest in this case are the symptoms of torticollis associated with bulbar paralysis in the patient and in the animals, and the predominance of lesions in the medulla and pons.

Three other cases were studied; in these torticollis came on insidiously without previous attack of encephalitis, and was of the usual type. There were no additional points of interest, and therefore detailed histories are superfluous. The salt solution suspension of the swabs from the nasopharynx of these patients was injected intracerebrally into eight rabbits. The findings are summarized in the Table.

SUMMARY OF RESULTS

The cases studied are representative of clinical types of torticollis. The ages of the patients ranged from thirty-three to fifty-seven years. Six were men and two women. In two the movements of the head developed after attacks of encephalitis; in one, apparently as a part of a mild form of bulbar encephalitis; in one, stereotyped myoclonic movements of the head followed what seemed to be a mild attack of wry neck; in four the condition developed insidiously, without apparent cause. The symptoms in all were more or less modified by external influences, and disappeared during sleep. Voluntary effort modified only slightly and temporarily the peculiar impulses. In one, mental strain was considered an important etiologic factor. A history of acute attacks of tonsillitis, sore throat, or influenzal infection was not given in any case. All the patients, however, had well marked evidence of focal infection in tonsils, teeth, or pharynx,

from which material for study was obtained. Removal of tonsils and infected teeth, and the continued use of vaccines made from strains isolated from the brain of positive rabbits was followed by the disappearance of abnormal hyperemia, secretion, and the characteristic streptococcus from the throat, and, in two cases at least, by temporary improvement in symptoms. The stereotyped movements of the head in the patients studied varied in duration from six weeks to four years, and were slowly progressive in character.

Blood-agar-plate cultures, made directly from the suspensions of material from the throat, tonsils, or teeth, yielded a predominance of colonies of green-producing streptococci in each of the eight cases studied. Hemolytic streptococci were not encountered, except in one patient who was recovering from an attack of acute tonsillitis at the time of study.

A summary of the results in each case following intracerebral inoculation of material known to contain streptococci is given in the Table. A high incidence of abnormal movements of the head occurred in each instance.

Of the seventy rabbits injected intracerebrally with suspensions or cultures of the streptococcus from the eight patients, forty-two developed peculiar tic-like movements, ten rhythmic movements, and twenty turning of the head; tremor developed in twenty-eight, hyperpnea in twenty-one, spasms of abdominal muscles in five, spasms of other muscles in ten, paralysis in twenty-two, nystagmus in eight, and lethargy in four (Table). Forty-eight died, a mortality of 69 per cent.

Cultures were made from the brain and blood in fifty-eight animals in this series. A non-encapsulated green-producing streptococcus, very similar in each case and closely resembling the one isolated in epidemic hiccup,⁽⁷⁾ was isolated from the brain in fifty-nine and from the blood in thirteen. The staphylococcus was obtained in only five and three instances, respectively, the hemolytic streptococcus once from the brain, and *Bacillus coli* from the brain in three and from the blood in four. The duration of the experiments in the animals yielding the streptococcus in the brain ranged from one to six days; in those that were negative, from seven to 193 days.

The agglutinating property of five of the strains was tested against my anti-poliomyelitis and anti-encephalitis serums, types I, II, and III pneumococcus serums, and normal horse serum. Three were agglutinated specifically by the encephalitis and poliomyelitis serums; the others were not agglutinated by any of the serums. The fermenta-

tive power of four of the strains for carbohydrates was tested. All fermented glucose, lactose, saccharose, and raffinose, and one fermented inulin, whereas mannite and salicin were not affected.

Filtrates of nasopharyngeal washings, of extirpated tonsils, or of emulsions of the brain of positive rabbits representing four cases, were injected intracerebrally into twelve rabbits. Nine remained free from symptoms, whereas three developed peculiar movements of the head and other symptoms of encephalitis. One of these recovered; the others died six and eight days, respectively, after inoculation. Characteristic lesions were found in both, and the streptococcus was isolated from the brain in one.

Seven rabbits were injected intravenously. In three, all of which were given sterile horse serum subdurally, localization of the streptococcus occurred in the brain, and in two of these, torticollis developed, and typical microscopic changes were found. In none of the four that remained free from symptoms were microscopic lesions demonstrable. Three rabbits inoculated in the anterior chamber of the eye with the streptococcus, and subdurally with horse serum, remained well and free from lesions.

THE SYMPTOMS IN ANIMALS

Two main types of symptoms referable to the head developed. In one, stereotyped movements of widely variable degree and kind were the dominant feature; in the other, abnormal positions of the head due to tonic rotary spasms of the muscles of the neck were the striking picture. Usually these types remained distinct from the onset, although not infrequently the latter developed after the peculiar tic-like movements subsided (Fig. 1). All grades of severity were noted, ranging in the former type from slight rhythmic movements of the head, resembling a true tic, to markedly stereotyped myoclonic contractions, producing most unusual pictures, and in the latter, from slight turning of the head to marked torsion, in which the head was rotated to an angle of from 45 to 90 degrees. In some the eyes were also rotated (Figs. 1 and 13); in others, the ocular axis was unaffected (Fig. 14). The animals often lost their balance, and tended to roll over and over, always toward the side to which the head and eyes were rotated. In these cases, the head could be forced into normal position, but immediately on release resumed the abnormal position. The intermittent abnormal movements were often greatly modified by external influences and disappeared during sleep. The tonic rotary spasm, on the other hand, was not susceptible to great

modifications and did not cease during sleep. Complete anesthesia, however, caused relaxation of the constant contractions of the muscles, and the head could be rotated readily and equally in all directions, even many months after the onset of symptoms. Contractures of tendons or bony ankylosis was not observed in a single instance.

Occasionally rotary spasm developed as a late manifestation in animals injected in the same way with material from encephalitis, epidemic hiccup, and chorea. Thus, rotation of the head and eyes developed in a rabbit (Fig. 13) ten days after an attack of spasms of muscles following injection of a strain from epidemic hiccup after



FIG. 13. Marked rotation of the head and eyes in a rabbit ten days after an attack of myoclonic encephalitis following injection of the streptococcus from epidemic hiccup after two animal passages and thirty-six rapidly made subcultures.

it was passed through two rabbits and thirty-six rapidly made subcultures. In the rabbit shown in Figure 14, rotatory spasms of the muscles of the neck, but not of the eyes, developed one month after an attack of chorea following injection of a chorea strain in the sixth rapidly made subculture.

The data in the Table do not adequately express the truly remarkable symptoms observed in this series of experiments. Even in the animals that died early from meningo-encephalitis, unusual movements of the head were often a striking picture; and in those that survived the acute attack, the stereotyped movements persisted often indefinitely in animals that were well otherwise. Not infrequently, these animals became abnormally sensitive to external stimuli in that they seemed to wish attention. On repeated occasions, teasing, such as

allowing them to smell carrot, of which they were very fond, and then taking it away, mere entrance into the room without opening their cage, tapping the nose and pulling the "whiskers", resulted in attacks in which after a series of exaggerated stereotyped myoclonic movements they would throw themselves over, often striking the walls of the cage. Stroking them and allowing them to eat carrot usually sufficed to quiet them. Long observation from outside of the room revealed the fact that these tantrums occurred only when the animals were aware of our presence.

Aside from an occasional animal with unusual resistance, the symptoms produced appeared to be due to particular neurotropic properties of the streptococcus injected. Consistent results followed injection of the streptococcus from the same patient on repeated occasions, and completely negative results after the removal of foci and prolonged use of a specific vaccine. In some instances the stereotyped myoclonic movements in the animals resembled closely those noted in the patient. Characteristic symptoms followed intracerebral, subdural, or intravenous injection of the freshly isolated strain after as high as forty-four rapidly made subcultures and through three animal passages. There was often marked similarity in the symptoms in a series of animals injected with a particular culture.

The results in one case were entirely negative following injection of the streptococcus isolated from the surface of an impacted tooth, contaminated with the mouth secretions in its removal, whereas all of the animals injected with the streptococcus isolated from the pulp uncontaminated developed stereotyped myoclonic movements. Moreover, in the case of the patient who was recovering from acute tonsillitis, torticollis was not obtained following injection of the suspension of the tonsil pus, which yielded large numbers of hemolytic streptococci. The green-producing streptococcus from the platings, on the other hand, produced characteristic symptoms.

Notable evidence of change in the type of symptoms occurred with one strain. When first isolated, nearly all of a series of rabbits developed peculiar movements of the head without lethargy, whereas after many rapidly made subcultures, lethargic encephalitis was common and was followed by characteristic movements of the head. Loss of characteristic infecting power occurred after long cultivation, and after long residence in the brain of the animals, especially in those that were recovering and were anesthetized for examination. In these instances, the strains no longer produced diffuse

growth in glucose-brain-broth, but a granular growth, and on blood-agar they usually produced small, dry colonies surrounded by a narrow zone of partial hemolysis instead of a greenish zone.

Results such as in these experiments were not obtained with inert filtrates, sterile sodium chlorid solution, and broth, and only rarely following injection of similar material from sources other than spasmodic torticollis. Thus, only ten (about 9 per cent) of 116 rabbits injected with the streptococcus from thirteen cases of epidemic hiccup developed tic-like movements of the head; only one (about 1 per cent) of ninety-two rabbits injected in the same way with material from the throats of forty-nine patients with acute



FIG. 14. Moderate rotation of head, with normal position of eyes, in a rabbit one month after an attack of chorea following injection of a chorea strain in the sixth rapidly made subculture.

poliomyelitis developed this symptom, and only three (about 3 per cent) of 106 rabbits injected with material from seventy-seven normal persons (Table). These results are in sharp contrast to those in the experiments on torticollis, in which forty-two of seventy (60 per cent) developed this symptom. To my knowledge, torticollis has never been known to develop spontaneously in rabbits.

GROSS AND MICROSCOPIC LESIONS

Peripheral lesions in nerves in the affected muscles, and in the internal ear and semicircular canals were not found in the animals that developed rotation of the head. The character of the gross and microscopic changes varied greatly, depending on the duration of the experiment, the size of the dose, the virulence of the streptococcus,

and the individual resistance of the animals. In necropsies on the animals that died in from one to five days, marked congestion of the vessels of the meninges, slightly turbid cerebrospinal and ventricular fluids, and occasionally hemorrhages in the medulla and cervical cord were found; and microscopically, a variable degree of leukocytic and round-cell infiltration, depending on the duration of the experiment. In those that died in six or more days after inoculation, gross lesions, aside from a variable degree of congestion of the vessels of the meninges, were usually absent, even at the point of injection; and microscopically, localized areas of infiltration, chiefly by round cells, were regularly encountered. The reaction incited was chiefly perivascular. Edema and cellular infiltration were often limited to, and always more marked around, vessels than elsewhere. This was true in the pia as well as in the brain and cord substance. Neurophagocytosis and degeneration of ganglion cells were slight or absent. The walls of the blood vessels were usually free from lesions. Accumulation of leukocytes within blood vessels, and capillary thrombosis were rare. Localized hemorrhages and infiltration and areas of focal necrosis were relatively slight and were often adjacent to blood vessels showing perivascular infiltration. In the animals that developed rotation of the head, unilateral lesions, most marked in the medulla and pons, were often found. The demonstration of streptococci in the lesions in animals that succumbed early was relatively easy, whereas in those that had recovered and were anesthetized for examination, it was difficult or impossible (Fig. 15 *a, b, c, d*).

Sections were studied microscopically in thirty-two rabbits that died in from one to five days after inoculation. All showed a variable degree of leukocytic and round-cell infiltration. Nineteen of twenty-four which were injected intracerebrally six or more days before examination revealed typical perivascular round-cell infiltration. This was also true of the two that developed symptoms after intravenous injection. It was not found in any of the animals examined within five days after inoculation, in any of seventeen which were injected with inert filtrates, sodium chlorid solution, or broth, nor in any of fifteen uninoculated rabbits of the same groups as those inoculated with the streptococcus, and in only three of seventy-six that had been injected intravenously with streptococci from sources other than diseases of the central nervous system. The high incidence of round-cell infiltration in the animals that lived six days or more following injection of the streptococcus from spasmodic torticollis,

effectively rules out the possibility that it was of spontaneous origin. It was hoped that the microscopic study of sections of different levels of the cerebrum, brain stem, medulla, and cerebellum would reveal lesions that would make it possible to postulate the precise origin of particular symptoms, but unfortunately this could not be accomplished. There was no close parallelism between the degree of symptoms and the extent or exact location of microscopic lesions. In some animals marked symptoms were noted during life and only slight lesions were found after death, especially in those that had the spasmodic type of torticollis, and *vice versa*. The rapidity of the development of lesions before compensating mechanisms had time to

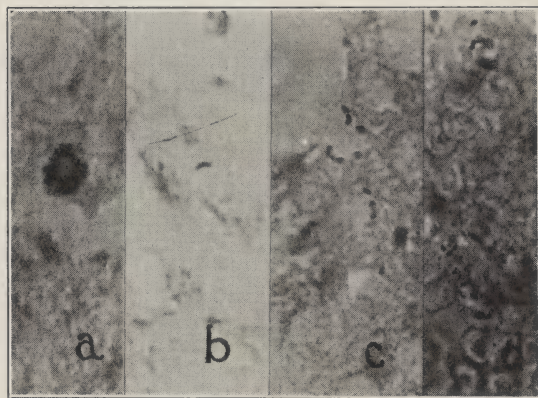


FIG. 15. Photomicrographs of the streptococcus in the lesions of animals. *a*, rabbit 2615; *b*, rabbit 3134; *c*, rabbit 3172; and *d*, rabbit 3300, injected one, seventeen, two and five days, respectively, before death.

develop appeared to be an important factor, a point supported by the recent work of Edwards and Bagg on cerebral localization. However, a study of the microscopic lesions in relation to the symptoms observed during life showed that peculiar movements of the head may be due to abnormal nerve impulses, the results of lesions of the nerve centers or paths in the subcortical region, in the basal nuclei of the pons, in the medulla, or in the cervical cord, corresponding respectively to particular groups of muscles affected.

The findings are in general agreement with clinical observations as regards the importance of lesions in the vestibular apparatus in disturbances of equilibration.(9) in my experiments lesions were not found in the labyrinth; they were always central. Rotation of the head in animals with lesions in the temporal lobe on the side toward

which the head was rotated, and no other lesions, supports the view that a vestibular center exists in the cerebral cortex. (1, 3, 6) According to Magnus and de Kleijn and Weiland, certain changes of posture and tonus of muscles may have been due to peculiar positions of the head. Magnus and de Kleijn demonstrated that the tonus of muscles was dependent on the position of the head in decerebrated animals, and Weiland found that extirpation of the labyrinth caused the head to turn to the side that had been operated on, and that the tonus of muscles and the posture were greatly altered by various positions of the head in normal animals as well as in those operated on. That streptococci sometimes accidentally cause vestibular disease is indicated by the fact that occasionally an animal developed marked vertigo and a tendency to roll over and over following intravenous injection of streptococci from foci of infection in patients with various diseases.

CONCLUSIONS

The results in these experiments indicate that spasmodic torticollis is often due to a streptococcus having specific localizing power for certain areas in the central nervous system. The presence of this organism in portals of entry long after onset may account in part for the progressive tendency of this disease. The removal of foci of infection when possible, the use perhaps of antiseptics to rid the throat of the specific streptococcus, and the application of specific methods of treatment, should do much to mitigate the distress incident to the affliction.

The fact that lesions were found in the central nervous system in animals that became abnormally susceptible to external influences, the symptoms often being greatly modified through the sense of sight, hearing, or smell, indicates that similar conditions in man may not necessarily be functional in origin, but may have an organic basis.

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ON THE DIFFICULTIES OF UTILIZING APHASIC SYMPTOMS IN THE LOCALIZATION OF BRAIN TUMORS: WITH A REPORT OF FOUR CASES WITH NECROPSY*

BY I. S. WECHSLER, M.D.

ASSOCIATE IN NEUROLOGY, COLUMBIA UNIVERSITY, AND ADJUNCT VISITING
NEUROLOGIST, THE MOUNT SINAI HOSPITAL, NEW YORK

Aphasia is generally defined as a disturbance in the psychic mechanism of speech; that is, it consists of partial or complete interference with the understanding, elaboration or expression of language in the presence of an intact peripheral speech mechanism. That a patient have aphasia it must be proved that his peripheral visual and auditory apparatus are normal and that his speech musculature and its innervation are unimpaired. Aphasia is further divided into sensory, motor and mixed. In the first there is supposed to be a defect limited to the inability to hear or see or understand language, although words can be uttered as a jargon or gibberish, and in the second there is supposed to be understanding of language and knowledge of what one wants to say, and at the same time inability to name objects, utter words or write them. More refined classification speaks of pure motor aphasia, pure sensory aphasia, pure alexia (word blindness), pure agraphia (inability to write), pure psychic blindness and a host of other schematically "pure" speech disturbances. Anatomically the division is made generally into cortical, subcortical and transcortical aphasia, and specifically, from the point of view of more accurate localization, into Broca's motor aphasia and Wernicke's sensory aphasia. The left third frontal convolution is supposed to be the seat of the motor aphasia and the temporosphenoidal lobe that of the sensory.

Thus far the definition seems to be very simple, the explanation fairly clear and the anatomical localization rather accurate. Unfortunately, most of the cases are not amenable to such sharp definition or classification. The general impression is that it is comparatively simple to state the kind of aphasia one encounters and to localize the lesion accurately. Experience shows this to be quite erroneous, and in recent years the whole subject of aphasia has undergone con-

* From the Neurological Service of the Mount Sinai Hospital, New York.

siderable revision. Thus Marie, based on his own work and that of Moutier, has attempted to revolutionize the whole subject of aphasia and to deny the existence of sharply localized speech areas, while Head has approached the subject from a psychological point of view. The attempts thus far have not materially clarified the situation.

The object of this paper, however, is not to enter into a discussion of the whole subject of aphasia nor to debate the question whether speech and intelligence go hand in hand, as Marie thinks, or whether they are independent of each other as Head contends. The purpose is to report four cases of tumor of the brain with necropsy findings and to discuss a few points in connection with the localization of the lesion by means of aphasia alone. This report may serve to reemphasize the fact that the accepted classification of aphasia and the generally assumed situations for the special types may lead to erroneous localization and even fruitless surgical intervention.

It should be stated further that aphasia is a symptom complex which is capable of careful analysis and that the term is too comprehensive for circumscribed localization. It is better, therefore, to say that the patient can understand what is spoken to him, read print or utter words, or understand what he sees, or read aloud or speak spontaneously or write words or carry out commands, etc., etc., rather than state that there is a motor or sensory aphasia.

It is true that it is frequently very difficult to analyze accurately the elementary speech disturbances encountered in brain tumors or other uncircumscribed lesions and especially where the general mental condition is such as to preclude accurate or detailed testing. In patients with more or less stupor, in young children whose speech contents are limited, in illiterate adults and in patients with generalized disease of the brain the difficulties are frequently insuperable. But even though a general impression as to the kind of aphasia is permissible in such cases, it is wiser to detail the meagre findings on which the impression is based.

Case I. H. R., age 46, married, female school teacher, highly intelligent and right-handed, was admitted to the hospital with the chief complaints of weakness of the right side of the body and difficulty in speaking. About five weeks before admission she began to complain of numbness and weakness of the right hand and soon thereafter of difficulty in finding the correct word to express herself. She gradually developed greater and greater difficulty in finding the proper word and the weakness extended to the arm and leg. She apparently understood everything that was said to her, could read her paper, carried on conversation, recognized the missing word

when it was supplied to her and generally made herself understood. There were no other disturbances; the past and family history were unimportant.

Physical examination: She had a right hemiplegic gait and attitude; there was a moderate Romberg sign, some incoördination of the right arm and leg and fairly marked disturbance in the performance of skilled acts with the right hand. There were no abnormal involuntary movements. The deep reflexes were livelier on the right than on the left, the right abdominal reflexes diminished or absent and there was a right Babinski. There was no hypertonus, but general weakness of the right upper and lower extremities. The sensation of pain, touch and temperature was possibly diminished on the whole right side of the body, including the face; vibratory sensation was present, position sense absent, and astereognosis present in the right hand. The vision and fields were normal, the fundi showed slight blurring of the margins of the discs; the pupils were equal and reacted promptly to light and convergence, there was no nystagmus and no ocular palsy. Corneal sensation was absent on the right. There was weakness of the lower half of the right face, hearing was unimpaired, the tongue deviated to the right. The heart and lungs were normal, blood pressure was 130 systolic, the urine normal, and the blood Wassermann negative.

Aphasic status: The patient understood all conversation, she was very alert and even grasped fine points of facetious remarks. She carried out ordinary commands, but seemed a little puzzled by very complicated ones. She knew the use of objects, could read and apparently understood what she read. She had difficulty in naming certain objects, could not find the proper words although she knew exactly what she wanted to say. She had the same difficulty with loud reading. She made mistakes in writing spontaneously or from dictation, but immediately recognized her mistakes. She could repeat the corrected words. She had a correct concept of speech, did not misuse words, employ a jargon or perseverate. She therefore had, with the exception of extremely slight impairment of the ability to carry out very complicated commands, an almost pure "motor" aphasia.

The diagnosis of probable brain tumor situated deeply in the post central (parietal) gyrus was made, although the possibility of softening of the brain was entertained. As slight choking of the disc gradually set in and within a few days the patient suddenly went into coma, a hemorrhage into the tumor was suspected and an exploratory craniotomy under local anesthesia, was decided upon. This revealed a large area of softening underlying a probable deep-lying, irremovable glioma. She came out of the coma after a few days, but she became incontinent, there was a bilateral Babinski, the hemihypalgnesia deepened, but there was no hemianopsia. The choking of the disc had meanwhile increased and there were retinal hemorrhages. She still understood everything, knew exactly what she wanted, begged that newspapers and books be read to her, but her fund of words gradually

became poorer and poorer. She lingered on for many weeks during which she alternately became comatose and alert, and after several such lapses she died in one of the attacks of coma. Necropsy showed an infiltrating glioma situated deeply in the right post-central area, in the parietal lobe, about three centimeters below the surface of the cortex and extending into the lateral ventricle, but not involving the precentral motor area or frontal convolutions.

Comment: The presence of hemiplegia with an apparently circumscribed motor aphasia without a history of localized convulsions should have justified the localization of the lesion in the motor area adjoining Broca's convolution, probably subcortically. Such a localization would undoubtedly have been assumed had it not been for the presence of astereognosis. As this is a sign of involvement of that part of the brain which lies behind the fissure of Rolando, that is, the post-central gyrus (parietal), the lesion was placed in that area. The astereognosis, therefore, which might well have been missed in a patient with a motor aphasia, was more important than the hitherto accepted localization of motor aphasia. This is of great practical importance, for had the craniotomy been performed over Broca's area the lesion would not have been found. Unfortunately in the present case the tumor was inoperable, but should it have been removable the wrong localization would have been a grievous error.

Case II. S. H. was admitted to the hospital March 22, 1922, in a stuporous condition. The history obtained was not altogether reliable, but his family stated that six weeks before admission they noticed a change in the patient's disposition. He became moody and slovenly and spoke very little. Two weeks later he is said to have had a "spasm" of the right side of the body, which then became paretic. He had no headaches. The condition is said to have improved for two weeks then gradually became worse again.

Physical examination four weeks before admission showed, in brief, slight paresis of the right side of the body and face, difficulty in carrying out commands, "word-naming aphasia," astereognosis (?) and choked disc. The diagnosis was subcortical temporosphenoidal tumor. Examination at the hospital: The patient was right-handed. Coöperation was very poor because of his stuporous condition. He had a definite right hemiplegia and a possible astereognosis. At times he carried out commands and uttered a few words, at others he could not be made to do either. There was choked disc of two to three diopters in the right eye and four to five in the left. He died on April 11, 1922, and necropsy showed a soft, hemorrhagic egg-shaped glioma wholly in the frontal lobe. Anteriorly the tumor was two centimeters from the frontal pole, laterally four centimeters from the surface and superiorly two centimeters from the surface.

Comment: The completeness of the aphasia, the probable aster-

eognosis, the early and marked choked disc (which is much less common in the frontal lobe tumors), the absence of signs and symptoms which are more often associated with lesions of the frontal lobe, led to the erroneous diagnosis of tumor of the temporosphenoidal lobe. But even allowing for the meager history and physical findings, owing to poor coöperation, there seems to have been, at least in retrospect, sufficient reason for suspecting a frontal lobe localization. The history of a unilateral spasm and of more marked word-naming aphasia should have suggested that. However, this case illustrates that aphasia alone was not sufficient to determine the localization.

Case III. M. G. was admitted to the hospital on May 15, 1922, with the history that ten months before he had a cold which was followed by difficulty in speaking. At that time he is said to have understood what was spoken to him but could not find the right word to express his thoughts. He was taken to a hospital and apparently got well in two weeks. Three months before admission he again began to have difficulty in naming objects and finding correct words to express himself. About six weeks before admission the right side of his body became weak and incontinence of urine set in.

Physical examination: The patient was right-handed. The salient features were, absence of choked disc, weakness of the left external rectus, paresis of the lower half of the right side of the face (supranuclear facial), right hemiplegia, with increased deep reflexes, absent superfcials and the Babinski sign, and speech difficulty most marked on its expressive side. The patient understood commands but could not speak. The diagnosis concurred in was tumor of the frontal lobe; and one observer committed himself to the definite localization of the tumor in the second and third left frontal convolution, on the surface.

Necropsy showed a large and firm temporosphenoidal lobe, which on section proved to be infiltrated by a large tumor. This did not extend into the frontal lobe, but did into the occipital. It was two centimeters below the surface and showed evidence of recent hemorrhage. The left ventricle was collapsed and pushed to the right. Microscopically the tumor was a glioma.

Comment: Owing to lack of coöperation on the part of the patient no detailed aphasic status could be made; but all the examiners were satisfied that there was an aphasia and stated specifically that it was motor in character. It may be seen, therefore, that the aphasia alone was not a reliable symptom in the localization.

Case IV. J. R., female, age forty-seven, left-handed, was admitted to the hospital in October, 1922, with the complaint that she suffered from headache and convulsions. The past and previous history were unimportant.

The present illness dated back to 1919 when one night she had a generalized convulsion. Since then, at the rate of once in two or three weeks, at times at longer intervals, she had attacks of the following nature: Her left arm and leg got stiff, she cried out, the left side of the face and left extremities twitched clonically and the eyes turned to the left. This attack lasted five minutes and was followed by exhaustion and severe headache. The attacks had become more frequent, but were uniform in character. She also had attacks of loss of consciousness, in addition to what seemed to be Jacksonian seizures, one attack lasting two days. She vomited a few times, but this was not projectile. She also complained of ringing in the ears, loss of memory, impairment of vision, inability to concentrate, difficulty in speaking (this came on late in the course of the illness), and weakness of the left side of the body.

Physical examination: The patient coöperated poorly. The fundi were normal. There was questionable weakness of the left external rectus. The lower half of the right face was weak, the tongue deviated to the left. The left deep reflexes were lively, the abdominals absent; there was a left Babinski sign and the left extremities were weak and flaccid(?). Sensation was not tested. The aphasia tests showed that the patient heard and understood spoken words and commands, she smiled at times when spoken to, she apparently could read and understand what she read and at times tried to carry out written commands. She could not speak.

A diagnosis of tumor of the right frontal lobe was made, she was operated upon, the brain was exposed but no tumor was found. She died and necropsy showed the following: A disc-shaped tumor, split longitudinally, was found lying between the two frontal lobes, beginning about three centimeters from the frontal pole and extending several centimeters backwards. The tumor could be shelled out from the left frontal lobe but not from the right. On sectioning the brain the tumor was seen to spring from and infiltrate the right frontal lobe, to grow toward the midline and extend into the left lobe. By pressure downward the tumor formed a trough in the corpus callosum, which limited the growth in all directions; the superior surface of the callosum was slightly infiltrated. The anterior horn of the ventricle was collapsed, the remainder dilated. The tumor measured five and one-half centimeters from side to side, six from above downward and seven from before backward. Macroscopic and microscopic diagnosis: infiltrating glioma of the right frontal lobe extending interfrontally.

Comment: In this case the left Jacksonian convulsions led to the diagnosis of tumor of the right frontal lobe and the aphasia (the patient was left-handed) confirmed the localization. But both the focal epilepsy and the aphasia, which was marked on the expressive side, pointed to a cortical lesion. Operation was performed in the belief that the motor area and Broca's convolution were involved,

while necropsy showed that neither was the seat of the lesion. It would be idle to contend that there may have been microscopic invasion of both areas, because from the clinical point of view the localization was not quite correct, the operation was fruitless and the symptoms were unreliable.

DISCUSSION

We have hitherto been accustomed to speak of aphasic speech disturbances in more or less schematic terms, but it has become increasingly evident that we cannot fit clinical pictures into theoretical schemes. Detailed examination, moreover, shows that "pure" speech disturbances are extremely rare. In case of tumors of the brain it is frequently very difficult to make aphasic tests because of the mental stupor. The lesion is usually not sharply delimited when a definite diagnosis of tumor can be made. But just because of this it is necessary to ascertain the exact details of the speech disturbance.

In general it is doubtful whether one can speak of circumscribed speech centers or accurately localize tumors (except in rare cases) by means of aphasia alone. The arbitrary divisions into pure motor (Broca's) and sensory (Wernicke's) aphasia or even cortical, sub-cortical and transcortical are more of theoretical than practical value. The above cases bear out some of these statements. All that can definitely be stated is that a tumor giving aphasia is nearly always situated in the left side of the brain in a right-handed individual. Speech disturbances may be encountered in frontal lobe neoplasms, in tumors in the lower part of the motor cortex, in temporosphenoidal lesions, in new growths low in the parietal lobe, in those involving the island of Reil, or the "quadrilateral space" of Marie. It should also be stated that lesions in those areas sometimes do not give any speech disturbances.

To add to the difficulty of localization of tumors of the brain by means of aphasia, there are distant or neighboring symptoms and late signs which may mask the clinical picture. Very early, however, in the course of the new growth, and it is only the early signs which are valuable in localization in any area, accurate testing may serve to bring out more or less definite disturbances. And in general it may be said that those which have to do with the emissive or expressive side of speech are more significant of lesions situated in front of the Rolandic fissure, while those which have to do with the receptive or perceptive or cognitive side of speech are more apt to be situated in

back of the Rolandic and below the Sylvian fissures. Aphasic disturbances, however, become extremely valuable for localization when accompanied by other signs and symptoms which in themselves are not sufficient for accurate localization. Thus, for instance, a focal (motor) epilepsy and aphasia would definitely place the lesion in the pre-Rolandic (frontal) area, while a hemianopsia or hemianalgesia and aphasia would point to a temporosphenoidal localization. But there may be exceptions even to these statements.

1291 Madison Avenue.

EPIDEMIC (LETHARGIC) ENCEPHALITIS *

A CLINICAL STUDY OF THIRTY-FIVE CASES GATHERED DURING THE RECENT EPIDEMIC, INCLUDING SOME RETROSPECTIVE CASES

BY MAX H. WEINBERG, M.D.

ASSISTANT NEUROLOGIST, WESTERN PENNSYLVANIA HOSPITAL,
PITTSBURGH, PA.

During recent years, since the epidemic of encephalitis has invaded practically the entire world, and established itself as a nosological entity, very much has been written on this subject, and one hesitates a great deal before undertaking the work of reporting such cases. But, this disease being so new, if only in conception, it is evident that what little light can be shed on the subject, what little added information, or even corroborative data one may contribute, justify the efforts of both the author and the reader.

The material reported upon in this article comprises thirty-five cases in all. Twenty-six of these cases were observed in the hospitals, mainly in the Western Pennsylvania Hospital, and I could follow most of them from their early inception or at most only a few weeks after the onset of the disease until their discharge, or death. Nine of the thirty-five represent retrospective cases in which there is not a vestige of a doubt as to the correctness of the diagnosis. All doubtful cases were kept out of consideration.

Reëxamination of as many patients as it was possible to trace were made from one to two and a half years afterwards, and whenever it was not possible to observe the patient, a report was obtained from either the patient, his friends, or his family physician. As a result of this follow-up work, the report also includes residua and sequelæ of a majority of the cases under consideration. This is especially valuable owing to our inability earlier in the course of the epidemic, say in 1919, and 1920, to prognosticate this condition correctly. It is only comparatively recent that reports on the sequelæ commenced to appear in the literature. Most of the authors strike a pessimistic note, and justly emphasize the seriousness of the condition from the standpoint of sequelæ. Thus, Bing and Staehelin (1) report on the sequelæ of eighty more severely affected patients who

* From the service of Dr. W. K. Walker.

have lived more than three-fourths of a year after the onset of the disease, only twenty-four of whom were cured. Grossman (2) reports that only ten cases out of ninety-two recovered fully, fourteen were functionally well, two remained stationary, and four showed some improvement. Sixty-two showed evidence of a more or less progressive involvement of the central nervous system, fully two-thirds of them showing a clinical syndrome that closely resembles that of paralysis agitans. While my series does not show such a preponderating number of Parkinsonian cases, yet the residua and particularly the sequelæ are sufficiently numerous and grave enough to cause one to consider this malady in a very serious light. Not only are the more grave cases apt to result in serious crippling of the individual, but even the so-called mild cases, such that have not been recognized during the disease and were ambulatory throughout the sickness, are apt to develop a serious sequel later.

There was no attempt made to classify the cases on account of my belief that all the classifications are forced and arbitrary. The disease attacks almost any part of the nervous system—although it seems to have a predilection for the basal ganglia and the floor of the fourth ventricle—and the symptoms naturally are an expression of the part of the nervous system involved. We may thus see a so-called choreic form of the disease go through stages of paralysis, myoclonus, and eventually terminate in a paralysis agitans syndrome. A case like this would have to be put under four different headings. I, therefore, described each case and arranged the symptoms in tables showing their frequency and proper relation.

In considering the material presented, one is struck by the greater frequency of certain symptoms and groups of symptoms. While each individual case presents a diagnostic problem more or less difficult of solution, yet there are certain clinical features occurring rather commonly, which should make one suspect the condition, if not make a definite diagnosis. Barker, (3) speaking about the spinal fluid in encephalitis, says: "In our experience, a cell count in the cerebrospinal fluid of from 10 to 100 small mononuclears, along with a positive globulin reaction with negative Wassermann and negative bacteriological smears and cultures is, at the time of an epidemic of encephalitis, strong corroborative evidence of the existence of the disease in a patient in whom the process is for any reason expected to exist." I would add that any time during the winter months—this was at least true during the last three winters—an acute infectious condition which persists and presents certain puzzling symptoms of

either an organic neurological or nervous character, should make one think strongly of encephalitis. The symptoms that should make one suspicious are, in the order of their frequency: alteration of reflexes, pupillary changes, paralysis, lethargy, diplopia, indications of pyramidal tract irritation or involvement, tremors, nocturnal delirium, and Parkinsonian symptoms. There are a host of symptoms of lesser frequency which occur often enough to aid one in the clinical diagnosis, but they may occur late in the disease when the diagnosis is well established, and can only serve as corroborative evidence or as mere neurological curiosities.

The examination of the spinal fluid is undoubtedly the greatest laboratory aid that we can summon in our study of these cases. The European observers still maintain that the spinal fluid shows as a rule no change. Thus, Howell (4) in the recent edition of *The Oxford Medicine* says under the heading *Cerebrospinal Fluid*: "This is found to be often under increased pressure, but as a rule presents no marked abnormality, an important point in the differential diagnosis. As a rule the cell count is not increased; occasionally there is slight pleocytosis, but not more than twenty to thirty cells per c.mm. are usually found. These as a rule are lymphocytes. Occasionally a trace of globulin may be present and also the Fehlings reducing power may be raised." This view is erroneous. The contrary is true. It is only seldom that the spinal fluid shows no change, and in those rare cases where there is no change in the cellular content and fluid reactions, there is at least increased pressure. Of the twenty-six acute cases in my series only three had a negative globulin reaction. The same is true about the cell count. While it is seldom as high as in meningitis, yet it is quite often higher than twenty or thirty. The cell count may even be very high.

ANALYSIS OF SYMPTOMS

Reflexes. Alteration of reflexes was noted in thirty-three cases, 93 per cent. This is by far the most frequent symptom, and while it is not being discussed much by various observers, probably on account of its being so common in neurological and psychoneurotic conditions, yet it deserves consideration. The outstanding characteristic of the alteration in reflexes is the inconstancy and inequality. Thus, one day some of the reflexes may be increased, and others diminished, and the very next day they may change their character and be just the opposite of the previous day.

Usually the reflex changes persist for a very long time. All the

retrospective cases and those of the acute cases that were reëxamined showed some abnormality of reflexes. Like irregular pupils, this is to be borne in mind as a residual symptom whenever syphilis is suspected in a patient presenting himself for examination.

Pupillary Changes and Paralysis. The two next most common symptoms in this series are pupillary changes and paralysis. Twenty-four cases, 69 per cent, showed pupillary changes. The pupillary changes vary from mild irregularities and sluggish reaction to light, to complete dilatation and static pupils. The changes may occur early in the disease or late.

These pupillary irregularities may clear up soon, or persist indefinitely. Eight out of my nine retrospective cases, and all the cases that I had a chance to reëxamine, showed pupillary changes from one to two years after the onset of the condition. Some may show improvement, but usually they remain the same as they were during the acute stage. This is a point well worth bearing in mind, since so many of these patients might be mistaken for syphilitics. This especially applies to cases that show residual paralysis as so many of them do present. The neurologist now has to consider the possibility of either a chronic encephalitis or a residual state of the condition, before condemning the patient as a luetic.

Paralysis or paresis was equally as common as the pupillary changes. The one distinctive feature about the paralysis is its migratory character as far as various parts of the body are concerned and its comparatively rapid disappearance. MacNulty (5) pointed out this feature early in 1918 and it still remains true. The paralysis may be moderate or severe, of short duration or persistent for a long time. It is but rarely associated with atrophy. Usually the cranial nerves are more often involved, but the limbs are frequently also involved. There may be monoplegia or hemiplegia, or the limbs may be involved in succession. The paralysis usually clears up before the patient leaves the hospital, and when it does persist it is usually only a palsy. The most frequent residual palsy is of the face or the tongue. Only one of my acute cases and two of the retrospective cases showed a marked residual paralysis.

Lethargy. The lethargy is the next frequent symptom encountered in this group—twenty-three cases, 66 per cent. The lethargy usually comes on early, but may appear later. Frequently it passes into stupor or even coma. There may be lethargy during the day, and delirium at night. This is especially true early in the disease. The two states may also be alternating, stages of lethargy



CHART I

SHOWING THE RELATIVE FREQUENCY OF SYMPTOMS IN THIS SERIES OF CASES

and delirium. An interesting feature about the occasional persistence of the lethargy is brought out in case 21. The mother reported at the reëxamination of the patient over two years after the

onset of the disease, that whenever he is very tired he may sleep for thirty-six hours at a stretch.

The delirium when present is usually of the infectious delirium type, namely, occupation delirium. The school girl talks to her teachers or classmates, the nurse who had war experiences lives through the war scenes, and the mill worker argues with his foreman. Ten cases out of the twenty-six acute cases showed delirium. The delirium seldom persisted after the first four weeks of the disease.

Diplopia. Diplopia occurred in eighteen cases, 51 per cent. This is believed to be a much more common symptom than is indicated in this series. It usually occurs early in the disease, and at times at the very onset, but it may occur as late as the eighteenth to the twentieth day. In case 3, the diplopia developed on the nineteenth day, and in case 24 on the twentieth day. In this last case the diplopia was of great help in corroborating the diagnosis of a myoclonic type of encephalitis which up to that time was in doubt.

Tremor. This was as common as diplopia in this series, eighteen cases, 51 per cent, having had this symptom sometime during the course of the illness. The tremor may be coarse or fine, slow or rapid, rhythmical or irregular, limited to one part of the body or generalized over the entire body. The tremors are not as persistent as some of the more severe symptoms. In the Parkinsonian type of case, the tremor of the hands is very characteristic, although the so-called pill-rolling movement is absent. Intention tremor is rather rare and occurred in but one case.

Babinski Sign. Sixteen cases, 46 per cent, presented this sign. This is a higher percentage than is usually reported. Alexander and Allen (6) in their analysis of 100 cases, report but seven cases exhibiting this reflex. Barker (7) merely mentions—"In a few, Babinski sign has been present." Other observers also speak of the rarity of this sign. But, if the cases are studied and observed carefully from day to day, it is found more often. Usually it appears later in the disease, except in cases that show hemiplegia early. When the Babinski occurs without paralysis, it disappears in a short time. It may also alternate from one foot to the other. Cases that remain paralyzed to any extent retain this sign for a long time. Thus, cases 7 and 32 showed a Babinski two and a half years after the illness, and case 33 one and a half years after the illness.

Öppenheim and Chaddock signs occurred in but few cases that showed the Babinski sign. A curious feature was observed in case

18. She never had a Babinski or a direct Oppenheim, but in the third week of her illness she had a crossed Oppenheim from right to left which changed in four days and was obtained left to right. This in turn disappeared in a few days.

Parkinsonian Symptoms. These appeared in fourteen cases, 40 per cent. This syndrome received more attention in the literature than any other symptom or group. This is due to the frequent invasion of the striatal system. Most of the observers report about the same frequency in this condition. Bing and Staehelin (1) reported thirty-six cases in their series of ninety-seven, Grossman (2) about forty out of ninety-two. House (8), however, reports but 11 per cent in his series of ninety cases.

Not all the cases that show Parkinsonian symptoms during the acute stage remain so, or progress. Of the fourteen cases, six remained stationary or improved slightly, one died, four recovered fully, and three became progressive, definitely marked Parkinsonian cases. Of the three, one, case 34, had a very mild attack of encephalitis which was not recognized until two years later when the patient presented herself with a fairly marked Parkinsonian syndrome. Only close questioning brought to the patient's attention the fact that she had a slight cold which kept her in bed only two days. At that time she had diplopia, blurred vision and headache. An ophthalmologist fitted her out with glasses and an internist diagnosed the condition as neurasthenia. The patient improved in about three weeks. Fifteen months later she was operated on for uterine trouble and soon after that she developed stiffness and tremor in the left arm.

Since I collected this material, I saw two other cases of post-encephalitic Parkinsonian syndrome in whom an anesthetic evidently precipitated the condition. It would seem that the intoxication resultant from the anesthetic is sufficient to either light up a latent infectious process or to bring about a degeneration of the diseased striatal cells that otherwise might have eventually recovered.

The invasion of the striatum may be mild or marked as is evidenced by an occasional segmental involvement. Case 7 showed involvement of the right hand and arm only. On the other hand, case 8 showed from the very beginning an extensive involvement of both the paleo- and neo-striatum, especially the latter. His choreic symptoms disappeared at the end of a week and the Parkinsonian symptoms were more marked until improvement set in, but about two days before his discharge, he developed a spasmodic tic of the left face and shoulder, which later spread to the entire left side of the body.

In accordance with Hunt's conception of the functions of the striatum, this patient must have suffered permanent damage in the neostriatal structure, while the paleostriatum recovered fully.

Cases 11 and 33 represent the juvenile type of Parkinson's disease, a sequel of encephalitis.

An interesting feature of the Parkinsonian cases is the festinating gait in moving the eyes. Sachs (9) terms it the cog-wheel movement of the eyes. On following an object moved laterally, the eyes move in jerks with definite stops and starts instead of the normal continuous and uninterrupted eye movement of the healthy individual.

Radicular Pains. Kennedy (10) quotes Abrahamson as having reported fully 60 per cent of his cases of encephalitis that had this symptom. In my series, thirteen cases out of the twenty-six acute cases, 50 per cent, presented this symptom.

I am making an exception in computing the percentage on the basis of the acute cases only, because the retrospective cases were examined about two years after their illness and the history of pains is not sufficiently reliable.

In six cases, this was the initial symptom and caused the patient to seek medical attention. In case 9, this was very characteristic and graphically described by the patient who dreamt that "somebody twisted his neck off," and on waking felt severe pain in the occiput and back of neck radiating downward. The pain would radiate over a nerve distribution such as from the neck down to the finger tips, or a girdle sensation would result in the abdominal area. In case 15, the radiation was upward from finger tips on left to the shoulder. In case 4, the pain was experienced in the kidney region and radiated anteriorly and posteriorly in girdle fashion. The pain, the only symptom at first, was so severe, that a diagnosis of nephrolithiasis was made. Only after ureteroscopic and X-ray examinations proved negative, neurological opinion was sought and the diagnosis cleared up. Usually the pain was limited to one area, but one case, 18, had shifting pains all through the course of the disease, practically covering the entire body.

An interesting feature is the fact that in the four acute cases that developed myoclonic movements during the disease, the radicular pain preceded the onset of the hyperkinetic phenomena, and in three cases the movements were limited to the area thus involved. Sicard and Kudelski (11) report four cases of acute myoclonic encephalitis in which the disease was ushered in with severe neuralgias, and lancinating pains. It would seem that the infection was either not

severe enough to invade the parenchyma of the cord and limited itself to the roots only, or that this particular virus had a predilection for the roots.

Headache. Headache is described as one of the common symptoms in this condition. Alexander and Allen (6) report 28 per cent in their analysis of 100 cases. Barker (7) merely mentions headache as a common initial symptom reported by English observers. Howell (4) also speaks of headache as a prodromal symptom. Thirteen cases, 37 per cent, in this series complained of headache. In nine of the cases it was either the initial symptom or the outstanding initial symptom. In case 3, the headache preceded the onset of the disease by two months and was persistent all through the illness. In one case the headache developed four days after the onset which was gastrointestinal in type. In case 32, the headache came on with the development of myoclonic movements, and kept up off and on for two years. On the other hand, in case 4, the mildest in the series, the illness started with a severe headache which lasted two days and disappeared with the onset of abdominal pain and myoclonic movements of the abdominal muscles.

The headache is severe and refractory to treatment. It may be limited to one part of the head or be general in character. In case 2, the complaint was more of a sensation of a tight band-like feeling around the head which persisted all through the illness, and even two years later the patient complained of the same disturbance coming on after eating.

Rigidity. This symptom was present in twelve cases, 34 per cent. The rigidity is usually of the Parkinsonian type. Frequently there is rigidity of the neck muscles in addition to the limbs, and it is difficult to determine in such cases whether it is due to meningeal involvement or not, but, the finding of rigidity eighteen months after the acute stage, as in case 33, would indicate that it is dependent on striatal disease rather than meningeal.

Sometimes the rigidity is extreme as in case 4, in which case the arm reflexes could not be elicited on account of the extreme rigidity. Three cases gave clear evidence of both meningeal and Parkinsonian rigidity. The persistence of the rigidity depends on whether the other Parkinsonian symptoms are progressive or stationary.

Rash and Desquamation. Little mention is made by observers of these very important symptoms. Although MacNulty (12) in his exhaustive study early in the epidemic called attention to them and

described various rashes, these symptoms seem to be overlooked. In this series twelve cases, 34 per cent, had one or both of these symptoms sometime during their illness. Usually they occur early, but they may appear very late. In case 11, so typical a measles rash appeared during the seventh week of the illness that the intern diagnosed measles and was preparing to transfer the patient to the city hospital. The rash disappeared in twenty-four hours. In case 6, the rash was at first confined to the abdomen and was suggestive of rose spots, but later changed its character and spread to the arms and head. This was followed by desquamation. Quite frequently, desquamation occurs without a rash being noticed. It is usually a fine scaling, but in the soles of the feet and around the toes it may be more pronounced. In a doubtful case, either a rash or desquamation is of great help in determining the diagnosis.

Confusion. Eleven cases, 31 per cent, showed this symptom. It occurred usually early in the disease. Cases 8 and 10 exhibited this symptom for over a week, and at the end of that time had no recollection of their surroundings during the past week, although they did remember their thought contents and some fleeting transitory illusions.

Nystagmus. Eleven cases, 31 per cent, showed nystagmus. Holden (13) reports 32 per cent for the Mt. Sinai cases.

In case 20, a rotary nystagmus developed on the tenth day of the disease. The movements were thirty to the minute, and the eyes would move three-quarters of a circle from right to left. In eight days this cleared up completely, but four weeks later, a day before he left the hospital, he developed a lateral nystagmus. Usually the nystagmus was lateral only, but two cases had both lateral and vertical nystagms. Three of the retrospective cases showed nystagmoid twitching, and two of these had cerebellar symptoms. The nystagmus cleared up fairly rapidly.

Ankle Clonus. This symptom was present in eleven cases, 31 per cent. In six cases it was exhaustible, and three of these cases, 25, 29, and 32, exhibited the symptom three years later. In case 10, this symptom was not present during the acute stage, but it showed up at the time of reëxamination two years later. In case 7, there was patellar clonus as well. The ankle clonus persisted and was found on reëxamination two years later. In five of the cases there was also a Babinski sign along with the ankle clonus.

Atrophy. This occurred in nine cases, 26 per cent, including among them two cases that showed mild atrophy of the tongue two

years after the acute illness. The atrophy was never very marked. Usually the interossei were involved. Four cases, 12, 14, 22, and 24, had either atrophy of the interossei alone or in combination with forearm and thenar eminence muscles. Cases 1 and 20 had atrophy of the leg muscles. Case 29 showed atrophy of the muscles of the left arm. Usually the atrophy was improved before the patient left the hospital.

Twitchings. Fibrillary twitchings of muscles, mostly the nasolabial were noted in nine cases, 26 per cent. In two cases, 13 and 8, the twitchings were sufficiently marked to move a limb, the right foot in the former, and the right forearm in the latter. In three cases, 12, 22, and 24, there were twitchings of the general musculature. It seemed like a continuous wave of muscle movements without interruption. Two of these cases died, the twitchings appearing a few days before death, and the third one was desperately ill and later developed marked bulbar symptoms.

In discussing twitching movements, Hunt (14) ascribes them to either disjointed fragments of striatal motility, or a release of kinetic impulses in subsidiary centers of the pons varolii. Barker, (7) on the other hand, thinks that they are due to the disease involving the cell bodies of the lower motor neurons. It seems to me that the twitchings of the general musculature indicate a grave intoxication of the system as is evidenced by the fact that most of these patients pass into coma and die. Only one out of the three survived. Sicard and Kudelski (11) report that all but one of these cases that they encountered passed into fatal coma. As death in these cases is mostly due to bulbar involvement, it is probable that Hunt's view of involvement of subsidiary centers is correct.

At least in one case, 20, the twitching helped to prove the diagnosis. This patient was on the medical service, and because of marked urinary disturbance, albumin, hyaline, and granular casts, the leaning was towards a diagnosis of uremia, but when the marked twitching of labial musculature was pointed out, the diagnosis of encephalitis was finally accepted.

Euphoria. This symptom occurred in eight cases, 23 per cent. Most of the observers merely referred to this symptom as of frequent occurrence. Kirby and Davis (15) say: "After the passing of the lethargic or delirious phase, euphoria frequently arises and with it sometimes uncontrollable laughter with appropriate mood." Jones and Raphael (16) reported euphoria in one of their four cases. Hohman (17) considers it "one of the most striking symptoms."

The euphoria is not quite as marked as in paresis. It expresses itself more as a mild sense of well-being and lack of insight. It is characteristic of these patients to insist that they are not sick and that they ought to be allowed to go home. The patients reported in cases 13, 15, 25, and 26, in particular, insisted practically all through the illness that they were well. The last mentioned had only been sick about ten days, and while her condition was getting gradually worse and paralytic symptoms had just appeared, she succeeded in prevailing upon her parents that she was not sick and she was taken home at the time when her temperature rose to 104 degrees.

In cases 7 and 20, there was uncontrollable laughter. In the latter case there was at first depression, but three weeks after the onset the euphoria developed. In case 32 there was a peculiar admixture of laughter and sadness two years after the onset of the disease. All of these patients, with the exception of case 8, had marked cerebral involvement, and the euphoria is probably dependent on the same factor as is the euphoria in paresis, namely, disease of the higher cortical centers.

Hypotonia. This symptom, although practically not mentioned by most of the observers, was found in six cases, 17 per cent. One of these, case 31 was a distinct cerebellar type and the examination made fifteen months after the acute illness. In this case the hypotonia was definitely cerebellar in origin. The hypotonia is not quite so easily explainable, however, in the other five cases in whom it appeared during the acute stage of the disease. Case 5 was especially interesting on account of the fact that the entire body musculature was extremely rigid, catatonic, with the exception of the left hand which was definitely hypotonic. This persisted for about a week. In case 24, hypotonia of both legs developed late in the disease, a few days before the patient died.

Vertigo. This occurred in but six cases, 17 per cent, in this series. It is reported much more often than this. It was an early symptom in all the six cases, and while very troublesome, it disappeared in a few days.

Illusions and Hallucinations. In five cases, 14 per cent, these were noted. They always manifested themselves during the early stage, the so-called irritative stage. They were mostly visual hallucinations, and the patient usually had somewhat of a vague idea of their unreality. Two patients, cases 6 and 17, saw great processions of soldiers passing by; two, cases 17 and 26, had auditory hallucinations during the first few nights of their illness. In the

former, the noise sounded as "shifting engines," but this may have been due to hyperacusis resulting from eighth nerve irritation, because she later had actual nerve deafness. The latter complained of "iron balls" rolling on the floor. There was no eighth nerve involvement in this case.

Two patients, cases 17 and 28, also had illusions. They saw grotesque faces in the flowered wall paper, and the latter also laughed at the monkeys he saw on the wall paper.

The hallucinations usually disappeared within a few days to a week.

Myoclonic Movements. Many observers have reported myoclonic movements in this disease. During the first year of the epidemic none was noted. Early in 1920, the myoclonic type appeared for the first time. When I encountered the first case, I was at a loss for a while and diagnosed it paramyoclonus multiplex, but in four days I observed pupillary changes and diagnosed the condition as epidemic encephalitis.

Five cases, 14 per cent, showed myoclonic movements. In one case, 32, both arms and legs were involved. In case 24, the legs and muscles of the back were involved. In case 19, the left arm was involved early and rather mildly, but two days later the patient developed very severe myoclonic movements of the right abdominal muscles. The movements were so marked that they would pull the head to the right side and even shake the bed. In case 4, there were movements of the left abdominal muscles.

The most curious and puzzling myoclonic movements were those observed in case 6. They were noted for the first time five weeks after the onset of the illness and came on with increased respiration. Synchronous with respiration, all the fingers of the right hand would move. After about four weeks, only the two middle fingers of the right hand still moved with respiration. This latter movement continued all through the patient's stay in the hospital, a period of six more weeks. Hamill (18) later reported a series of four cases in whom he observed the same phenomena. Hamill arrives at the conclusion that these movements are caused by involvement of a mechanism or center in the medula. This is probably true, since the movements came on with increased respiration, and thereafter were synchronous with it.

Hunt (14) ascribes the myoclonic movements to some infratriatal mechanism of the paleokinetic system; while Scott (19) reports a case with myoclonic movement of the abdominal muscles on both

sides, more marked on the right, that came to autopsy. The main lesion was localized in the left precentral gyrus.

Cerebellar Symptoms. In five cases, 14 per cent, cerebellar symptoms were noted. Two of these cases, 30 and 31, are in the retrospective group, and their symptoms may be considered as residual.

A curious feature is that all the five cases showed the cerebellar symptoms mostly on the left. *Adiadochokinesis* on the left was present in all the five cases. *Hypotonia* was found in two cases, *nystagmus* in two, and *pass-pointing* in two. The following symptoms were found once: *staggering gait*, *asynergia*, *disturbed speech*, *dysmetria*, and *pendulum swing* on taking the knee reflexes.

With the exception of the two retrospective cases, all the cerebellar symptoms disappeared at the time of reexamination, usually about two years after the acute illness. Mild mental symptoms, however, persisted in two of the three acute cases.

Meningeal Symptoms. These occurred in five cases, 14 per cent. In only one case, 25, were the symptoms found at the very onset. The patient complained of a very severe headache, and he had marked rigidity of the neck and bilateral Kernig sign. Immediately after the lumbar puncture, the meningeal symptoms cleared up. The fluid was under great pressure (see table I). It was more like a case of serious meningitis. In the other three acute cases, 9, 11, and 22, the meningeal symptoms appeared later in the disease and cleared up fairly rapidly, with the exception of case 22 that terminated fatally, in whom the meningeal symptoms were marked as were all his other symptoms.

I cannot give the extent or severity of the meningeal symptoms in case 29, a retrospective case, but I included it because the diagnosis of meningitis was made in the army, and it is fair to assume that meningeal symptoms were present.

Retention of Urine. This occurred in five cases, 14 per cent. Usually it occurs early, not later than the tenth day, and clears up in a few days upon the administration of pituitary substance in small doses. In case 22, this was of considerable help in establishing the diagnosis which up to the manifestation of this symptom was doubted and considered by others a case of hysteria.

Fundi Changes. In four cases, 11 per cent, fundi changes were observed. In only one case, 3, was a choked disk noted in the left eye, and a beginning choked disk in the right eye, during the third week of the disease. On account of this finding, the condition was diagnosed by the attending physician as brain tumor. When the

patient reached the hospital, our ophthalmologist, Dr. Carson, reported "only a paleness, slight evidence of sclerosis."

In case 2, there was peripillary edema of the right eye, and in case 19 there was definite blurring of both fundi, but this cleared up rapidly. In case 30, a retrospective case, the left fundus was almost white.

Reversed Sleep. Usually this is a symptom reported to be found in children only. Hofstadt (20) considers it so characteristic of encephalitis that this about justifies the present or retrospective diagnosis of the disease. Kirby and Davis (15) mention four cases in the reversal of the day and night cycle of hypersomnia and hyposomnia.

In four cases, 11 per cent, there was a reversal of sleep. Two were adults, one was an adolescent, and one a child of five. In two adults, cases 18 and 19, the symptom occurred during the acute stage of the illness and lasted but a few days. In case 21 the condition developed after the child left the hospital and it kept up for three months. In case 28, the condition came on about two months after the onset of the illness. This patient was lost track of, and the duration of the symptom could not be determined.

Psychoneurosis. This was evident in four cases, 11 per cent. In two cases, 15 and 17, the condition occurred during the acute illness. In one of them, case 15, it remained as a permanent sequel, but there was a complicating factor, namely, the patient being an ex-soldier, and the subsequent attempt to obtain compensation. I could never determine what part this factor played in maintaining the psychoneurosis. I am quite satisfied that it played no part in the development of it. He at first complained of a feeling of being torn apart and cried for help, continually repeating that "I cannot stand it." This condition kept up with but slight improvement for three months before the patient ever knew that he was entitled to government aid. But, after that, he became distinctly worse, was at the government hospital for psychoneurotics for over a year, and at the time of the reëxamination, twenty-two months after the onset, he was even worse. He then complained of digestive disturbance, constipation, dizziness, nervousness, and extreme restlessness.

(To be continued)

STATIC SEIZURES IN EPILEPSY: REPORT OF TWO CASES

BY KARL A. MENNINGER, M.D.

TOPEKA

Static seizures in epilepsy are characterized by Ramsay Hunt¹ as sudden losses of postural control, to be differentiated from the ordinary convulsion which represents the kinetic type of seizure.

His paper as abstracted in the JOURNAL OF NERVOUS AND MENTAL DISEASE should be quoted in part in order to justify the inclusion of the following case which I believe belongs in the same category.

"The static seizure may occur alone, as a dissociated manifestation of epilepsy. The loss of postural control is sudden and shock-like, the patient falling to the ground with abrupt violence in response to the law of gravity. This sudden plunge or drop is characteristic of this type of seizure and is not infrequently the cause of serious injury, especially to the face and head. It is quite different in its character from the usual fall of the epileptic in the convulsive attacks. In one case, both patellæ had been severely injured by frequent and severe *drop-seizures*. While the drop is sudden the postural relaxation is only of short duration, the patient rising almost immediately from the ground without assistance. The fall is usually associated with transitory loss of consciousness, which may, however, be very slight. In not a few instances there is scarcely any appreciable obscuration of consciousness. The fall is usually forward and is associated with a sudden relaxation or 'giving way' of the lower extremities. As a rule there are no convulsive manifestations, although the two varieties of attacks may be combined. In the type of seizure just described the postural relaxation is more or less general in character, the patient falling in a heap from complete loss of postural control."²

In the discussion Dr. L. Pierce Clark said he had never seen such

¹ At the 397th regular meeting of the New York Neurological Society in June, 1922.

² Hunt, Ramsay, J. "On the Occurrence of Static Seizures in Epilepsy," JOURNAL OF NERVOUS AND MENTAL DISEASE, Vol. 56, No. 4, October, 1922, pp. 351-352.

attacks in epilepsy, referring no doubt to Hunt's statement that he had observed these seizures only in cases of idiopathic epilepsy.

At the same meeting Dr. Dana reported a case which was evidently of the same sort. It was a boy who "would crumple up in the street or at home with instantaneous relaxation of all of his muscles. There might have been an unobservable period of muscular tonus first, but he could not see it. The boy simply dropped right down and got up again at once. The case was reported as true falling sickness."

I wish to present two cases conforming to the descriptions of Hunt and Dana of static seizures. In the first case the drop-seizures constitute one of three types of epileptiform manifestations; in the second case the drop-seizures are the only symptom. This corresponds with the statement made in the first sentence of the above quotation from Hunt.

Case Reports

Case 1. T1037 is a boy of four, referred to us by Dr. Mayer Shoyer. Family history is negative for any neurological, mental or epileptiform incidences except that both parents regard themselves as "nervous." The patient is an only child and there were no miscarriages.

Present Illness. The gist of the story is that at the age of two the child one day had a typical major convulsive seizure which recurred in six months, again in another three months and thereafter about every three or four weeks. There was the usual stages of tonic, clonic, stertor, sleep and malaise, but no aura. Vomiting occurred with every attack and also in paroxysms between times.

Shortly after the onset of the grand mal, typical petit mal began, described as "spells in which he loses himself just for a moment, stands perfectly still, stares vacantly straight ahead. He most always grunts in these attacks, but not in either of the other kinds." At the time of the examination the little patient was having all the way from none to 40 of these attacks daily. The irregularity was too great to strike any average.

Apparently *pari passu* with these grand and petit mal he began to have what his mother calls falling attacks of which her description needs no amplification:

"He'll be sitting on the foot-stool and suddenly drop like he was shot, and hit his head kerplunk, get right up again and be all right. Or he'll be standing up, pulling his wagon, say, and suddenly tumble over backward. (He falls forward or backward.) He'll bump his head, cut a gash, perhaps cry a little but more often just jump right up immediately. These don't come when he's standing still—he is usually moving, and when he pitches forward he often put out his hands to catch himself. This morning he fell forward as he sat at breakfast,

his head splitting the plate. He sat right up and went on eating. He just drops like he's shot."

Examinations: Physical, neurological, and laboratory examinations revealed nothing of particular importance in the consideration of the system of static seizures. A diagnosis of idiopathic epileptic syndrome, with grand mal, petit mal, and static seizures was made.

Case 2. W729, is six years old, the daughter of healthy parents aged thirty-five and thirty-two respectively and with excellent heredity. She has one sister two years younger who is also in excellent physical and mental health. Her mother has had no miscarriages nor have there been any other pregnancies.

There is positively no history of epilepsy or convulsions or what might be interpreted as equivalents in the family history, and this could be fairly accurately determined because the father is an undertaker and both his father and his brother are capable physicians.

This child was born in August by normal labor and was breast fed until the usual age. She learned to talk at twelve months and to walk by herself at sixteen months. At four and a half she had both chicken-pox and whooping cough but aside from this she had always been perfectly well.

Present Illness: When the child first began to walk it was noticed that she fell frequently. At first it was ascribed to her childish clumsiness but then it was noticed that she would fall without provocation at a time when her balance was well stabilized. As she grew older a more definite clinical picture could be defined.

The essential feature is still that *she suddenly crumples up and drops to the floor*. She does this under all sorts of circumstances, and very frequently. There may be little or no provocation but it is definitely known that sudden noises such as handclapping or even calling her by name are sufficient to make her fall down in a heap wherever she is standing or running. She always gets up immediately, shows no evidence of having been unconscious and complains of no pain. She is perfectly aware that she has fallen but makes no apology for or explanation of it.

Dr. Karl Robert Werndorff, orthopedist, formerly associated with Lorenz at Vienna and now of the Hatcher Clinic at Wellington, Kansas,³ examined the girl recently and pointed out that the falling seemed to take place at times as if there were a sudden failure of the thigh adductors. This was not constant, however, and there was no anatomical confirmation of the theory.

At one time the parents said there had been a tendency for her to fall toward the left but this had not persisted.

Examination: The little patient is an exceedingly attractive little child with no stigmata of degeneration either anatomical or physiological which could be determined objectively. There were likewise no hysterical stigmata. She is a sober-faced, serious-minded,

³ The case was seen by me with Dr. Werndorff, at Wellington, and is reported by his courtesy.

obedient child, calm and quite likable and self-possessed before strangers. Under no circumstances could she be regarded as what is connoted by the lay terms "nervous" and "high strung."

The most painstaking physical, neurological, serological and orthopedic examinations were entirely negative. To indicate the thoroughness with which we investigated the neurological data I may say that Fournier's gait tests, Babinski's five tests for asynergia, André Thomas' tests for dysmetria, tests in past-pointing and in coordination, careful dynamometric measurements, orthopedic test procedures, etc., were all carried out (without yielding any abnormal results). A few months previously she had seemed flatfooted but this had been spontaneously corrected.

The one exception to the thoroughness of the testing was in the matter of the spinal fluid which was not examined. The parents' blood sera gave negative Wassermann reactions.

Diagnosis: One seems obliged not only by exclusion but also by close parallelism of the clinical description to fall back upon Hunt's "static epilepsy." One might theoretically postulate an incomplete synaptic development but this would hardly explain the case so well as the proposed diagnosis. It has certain features which seem to be unusual, particularly the ease with which attacks were precipitated. As a matter of fact the history led me to expect them to be much more frequent than they proved to be at the time of the examination. It was only with difficulty that I convinced myself that it was possible to deliberately evoke a fall.

SUMMARY

Two cases are presented in which there occurred static or drop-seizures, corresponding to the description of Ramsay Hunt and others. They are to be regarded as an unusual manifestation of the epileptic syndrome. In one case the static attacks were the only symptom; in the other case they were associated with attacks of grand and petit mal.

MULVANE BUILDING.

TRANSLATIONS

EMOTION, MORALITY, AND BRAIN*

BY PROF. C. V. MONAKOW

ZÜRICH

(Continued from page 545)

VISCERAL-NERVOUS SYMPTOMS IN THE ADULT

The viewpoint of considering the neurosis as a protracted or faulty reaction of the individual to injuries to his personal vital interests, to unfulfilled (justified and unjustified) wishes*and hopes and so forth, cherished and ardently desired by him, concedes an enormous significance to the etiological, that is, to the genetic historical moment. The developmental, that is, the genetic moment, forms a viewpoint that plays a wonderful rôle in the total biological knowledge. This view assumes as its point of departure the successive urge to "become" of things, from the creative phases, from the temporal (historical), exactly organized construction of the nervous functions as a whole, and having regard, on a broad scale, for the constant reciprocal influences of the manifold impelling forces, both inner and outer. It points to the fact that all living creatures are in a progressive or regressive state of fluctuation. Moreover, for the origin of the neurosis, the biological moments are decisive; the quality of the germ, its intrauterine development, then the early education and experience in childhood (injuries to the vital interests even during the embryological period of development). In other words, the history of the evolution of the emotions remains under the constant influence of the same forces which have fashioned the organs and the nervous system. It is a question, then, of a true circuit. Pathological stimuli (poisoning, even autointoxication), diffuse and inflammatory processes early acquired (both in the meninges and in the cortex) play a special and certainly not wholly unessential, even if not decisive, rôle in the development of nervousness. However, they are not directly responsible for the origin of

* Authorized translation by Gertrude Barnes, A.B., and Smith Ely Jelliffe, M.D., of the authors *Gefühl, Gesittung und Gehirn*.

the neurosis, just as little as are other locally limited, acute cerebral lesions (for instance, foci of vascular nature, trauma, tumor, and so forth) or excessive demands on the brain (in the sense of mental or physical overexertion, without producing conditions of personal conflict). But all the above mentioned moments, which must not be underrated, may delay or hinder the solution of conflicts released by exogenous moments because, like the inferiority of the germ, that is, the anlage, they prevent the natural outlet for the nervous reaction to outrages to personal interests, and direct the different abnormal emotional tensions over a false path, and so on. Enough, we can conceive the neurosis and in large measure the psychosis as an unresolved phase in the struggle for the most important life-interests of the individual (self-support, maintenance of the race, at any rate of human society and community with God or nature, the world), a conflict which may be carried to inconceivable heights or be of long duration. It is a strong, protracted reaction on the part of the nervous system in the sense of a defense against injuries, usually from without (as they happen in daily life) or in the sense of a counter attack or struggle for precedence on the part of manifold, positive emotional values (power, possession, success), as a reaction which controls not only the world of feeling and especially the subjective causality, but also—through the mediation of the visceral nervous system—the inner secretions (defective circulation). Of course, through this struggle or “reaction,” the rule of conduct for the affairs, projects, and enterprises of the patient will be indirectly influenced in the most effective way, at all events as far as it is a question of the entirely personal side.

The neurosis may appear, as is well known, in manifold forms, and in every form there are very diverse combinations of symptoms, and also typical variations and phases. Here above all are characteristic the acute exacerbations in the shape of “attacks” or “crises” definitely limited as to duration. In the majority of cases of neuroses unmistakable accompanying somatic phenomena occupy the foreground, and without exception during the exacerbation (“seizures,” emotionally toned complexes).

The usual clinical classification gives, as you all know, the following forms of neurosis, which not seldom overlap one another: hysteria, neurasthenia, phobia, that is, the anxiety neurosis, the depressive conditions of hypochondriacal character, simple, temporary “nervousness,” and so forth. The outer insults which come under consideration either as essential or as favorable moments

where there is predisposition, either innate or acquired ("hystero-neurasthenic variations in the nervous system") are:

(a) Severe menace or danger aimed directly or indirectly at the individual existence or security (fear, care, bodily illness), whether in the form of a direct, swift attack (sudden attack, catastrophe), or in the form of a more stable situation filled with care and suspense.

Here belong:

1. With reference to the individual life: loss of freedom (imprisonment), economic ruin, plots against life, persecution, and so forth.

2. With reference to the interest of the race: disappointment in the selection of a wife, faithlessness of the loved one, brutal sexual attack in childhood, seduction, rape, attack on one's sexual honor, acquaintance with sexual perversity of all kinds, unfulfilled sexual wishes cherished ardently in secret, and sexual conflicts of many kinds, and so forth.

3. In social relations; violation of honor, slander, oppression humiliation, exposure, betrayal, disappointment, undermining of authority, loss of dearly loved friends, religious persecution, psychic crisis and trials, then catastrophes affecting the native country, war, devastating epidemics, and so forth.

Moreover, in the occurrence of a psychosis, an essential rôle may be granted to outrages on peaceful life, or the conflicts connected with it (real or even frequently of a delusional nature), even if one ascribes the main rôle to the "endogenous" moments and pathological processes in the central nervous system. In any case, it is a question not only of the threat to the most vital interests of the individual, in the immediate present, but especially of such in the *future*. Of course, to reiterate the insults just now detailed, if a psychosis, psychasthenia, or neurosis is produced, must work upon a nervous system shaken by manifold occurrences in previous life (accumulated poisonings by inner secretions, that is to say, possessing a tendency to disease, through diathesis, independent pathological cerebral processes, or as result of milieu or other influences of manifold sort. In these processes remembrances of similar earlier experiences (homophony) in the form of mnemonic stimuli play a powerful rôle determining the special nature of the symptoms. In every offense to the most vital life interests, a "conflict" is occasioned by the production or awakening of inner contrary stimuli (chemical defense of the body, ferments?), and the perplexed individual helps himself at this point—in his feeling of freedom—with many effective

or even ineffective weapons supplied him by nature; or he seeks his defense in flight, in concealment, in denial (ignoring) in death or in the pharmacopœia, that is, in narcotic means (alcohol and so forth), which, as experience shows, can allay the struggle with the secretions poured into the blood.

The behavior of the individual is the expression of the reactions produced through the psychic or bodily insult, that is the reaction of the central nervous system to the real or delusional outrages. The emotions (affects) released thereby cause, in my opinion, an increase or an arrest of the inner secretions (adrenalin? thyreoglobulin? [Oswald], etc.); they even create perhaps other "conflict ferments," from which result all possible further consequences for the health.

The psychic reaction, where there is menace or violation of the more vital personal interests, may, in healthy persons, and regularly does, assume manifold forms. Sometimes it assumes the character of a direct counter-attack, or of an impassionate preparation for such (subconscious latent phase), sometimes it consists in ignoring or in a passive opposition, that is, a true "inhibition" (defense against a surprise attack?), or in flight, evasion, hiding, eventually in wishes to disappear from the picture, or in malingering, the last of which conceals in itself a "retrospective wish in relation to facts."

In my experience, either the contestant takes up the conflict, or else he restricts his response to the more passive principles of defense (even to self-control), according to his momentary capacity for resistance, character, always according to the nature of the released affect, and also according to his comprehension of the situation at the time. The intellect also serves the defensive and protective emotions here and their respective defensive actions.

All the forms just now depicted of protection and defense we meet in the neurotic if he receives a psychic trauma, that is, he controls himself according to his education, emotional condition, and character, either through active return of the insult, or—and this is an especially frequently appearing phenomenon—he seeks to flee or postpones the defense—and controls himself silently—disguises his feelings, or lies. Through this control, the emotional tension created by the insult (anger, revolt, hate, pain, disgust, and so forth) is not usually lost; it becomes only latent, and disposed of otherwise (according to Freud, it is "repressed"; a conception which is not always applicable). The emotional tension presses in secret towards a discharge whereby—without control through reflection—it is projected in various emotional spheres and indeed to the very so-called

substratum of the emotion. Henceforth it exercises a harmful, regressive influence even over the inner secretions (patient is "poisoned"). This is besides a physiological factor, which contributes much towards the building of "emotionally toned" complexes. The distribution of the processes of excitement corresponding to the heightened affects comes to pass in seemingly unaccustomed ways which are not under the control of consciousness—it is a question of an instinctive elaboration—mainly in the subconscious. The prevented discharge of the emotional tensions leads to various disorders both of the psychic and of the bodily equilibrium (disturbed "health"). This *projection* (transplantation of the stimulus into an unaccustomed, seemingly foreign innervation sphere, and defense on the part of psyche; the "conversion" of Freud) may result:

(a) In the periphery, that is, in the material sense, as well in the sphere of the visceral as of the cerebrospinal central nervous system, and indeed (under a certain election still incomprehensible to us) in very definite, occasionally spatially sharply limited, if also not anatomically outlined bodily parts and apparatus, which are of significance for the indirect or direct maintenance of, or the economy of, life (hysteria).

These "projections into bodily matter" are sometimes direct consequences, in my opinion, of the naturally resulting, acute storing up, in every strong affective process, of the inner secretions (overflow of adrenalin, thyreoglobulin(?) or ferments still unknown to us). Whether one can perceive here a protective contrivance, I cannot decide, but it seems not impossible. The somatic disorders can relate to:

1. The circulation and heart action, respiration (the oldest and most vital activities maintaining life): tachycardia, extrasystole, vasomotor, tension, tachypnea, and so forth.

2. The organs of digestion (symptoms: diarrhea, vomiting, nausea, that is, impulse to eliminate the undesired material through the digestive tract), dyspepsia, obstipation, and so on.

3. The sexual and urino-genital organs (heightened or retained secretion and plethora of the genital glands, frigidity, or heightened desire, the retention of the urine, and so on).

About each one of these three visceral spheres a whole complex of dysesthesias (including Head's zones) and temporary repression of functions of all kinds (symptoms of hyperactivity and of paralysis) can be grouped. The various kinds of neurosis can be divided

very well in this way: neurosis of the heart, nervous dyspepsia, that is, neurosis of the stomach, and sexual neurasthenia.

However, repressions of innervation can appear in very similar manner within the cerebrospinal nervous system, the orientation system (that is, in connection with repression of the inner secretions and of an even psychic modification corresponding thereto). These disturbances may carry either the character of the excitation or of the paralysis (inhibition), that is, the so-called hysterical appearances of excitation and paralysis, which can carry a narrowly localized character (limited to very definite bodily parts; often to one side), and which can imitate all possible forms of organically produced, localized disturbances (even to convulsion and hemiplegia, monoplegia, paraplegia, and so on). They are definitely distinguished, nevertheless, from the organic disturbances, as much through the character of the "dissolution of the function" as through the nature of their onset. The more definite anatomical points of attack for the spatial hysterical stimulus and paralysis symptoms are unknown to us, just as is the nature of the sources of pathophysiological stimuli now in question (intoxication, electric, chemical or "mnemic" stimuli?), and whose closer study is beset by the greatest difficulties. One speaks of this in a clinical way usually of "psychogenic" forms of excitation and inhibition, whereby one seeks to characterize, nevertheless, only their source, in contrast to the roughly localized pathological-anatomical forms of functioning.

(b) Projection in the obscure world of the instincts, and in the subjective causality misled through the insult, that is, projection in the so-called instinctive thought (unconscious reflexion). Anatomically represented, this flooding with abnormal stimuli, especially with mnemic stimuli of many kinds and grades of development (experience and episodes from various periods in the past with their wider consequences) affects the cortex exclusively. This disturbance of equilibrium (now of a retarding, now of an irritative nature) represents a truly Protean picture of disordered, uncoördinated structures, even to the phantasms in confused conditions, and to delirium with eclipse of reality, and the apparent subversion of all logic. Here, first of all, belong the hysterical confusion and dream states. In these one often encounters mad activity of the instincts with corresponding excited modes of expression and with "passionate attitudes"; the activities exhibit here a very great variety of character (*grande hystérie*).

Of course the whole process here has the character of a defense, though it may be ineffectual or without plan, of the wish for liberation from the psychic condition insufferable to the individual (pain, anxiety, and so forth), thus of a kind of psychic expulsion.

Anxiety and the accompanying compulsive conditions constitute a well-known main type of these acute or intermittent conditions appearing in attacks: *phobias* (anxiety neuroses). I regard it as unquestionable that here again the inner secretions or the acutely altered blood composition play a part in these disturbances, and that it is not a question here merely of fixation, of "converted thought."

(c) Projection or transference in the logically built up subjective causality, in so-called reflexion. Every strong acute insult—no matter whether somatic or psychic in nature—does not only raise the affect to a certain intensity, but controls simultaneously (automatically) the subjective causality, that is, the inquiry for the cause. For every man, after he has apprehended the insulting stimuli spatially and temporally, the first question is: Where does it have its own source; what has brought it about (causality)? and the second: When and how can it be removed in the swiftest way (defense); what does it forbode for the future, especially with reference to the further prosperity of the personality suffering? The direction of thought just now indicated can, under pathological relations, set in directly as a "form of a convulsion"; it can return and periodically recur (perseveration of a narrowly limited intellectual circle). In this category brooding belongs primarily, built up, as it is, in external, logical form, but characterized by "egocentric" moments, and accompanied by obscure instincts. It is a meditation on the relation of cause and effect in regard to the stimulus or occurrence, as well as over the means to protect one's self from similar undesired experiences, and eventually from wider (secondary) consequences of them; that is, to ward off or to free one's self from present difficulties. Hereupon perplexity falls upon the patient; there arise all possible painful conjectures, misgivings, apprehensions, and so on, which may take the form of perseveration. For defense the strangest, often most unsuitable means, are instinctively chosen, often because of their relationships to earlier, impressive experiences. This form is regarded as real melancholy brooding.

Every neurosis erects itself on the three forms of "elaboration" (of highly affectively toned experience either not discharged or not satisfied) described in the preceding, and the elements of such "reaction modes" (forms of projection), alone or mingled with others, are encountered also in the majority of the psychoses. From

first to last these are defensive and protective reactions for securing positions of safety (mostly proceeding from false hypotheses), or finally of preventive attacks and enterprises.

To understand better the mode of functioning of the various experiences and occurrences menacing our emotional world, we take as our starting point a mature, sound man, who lives and works in general with a vigorous surplus of joy in life. Illusion (in the sense of daily life), that is, confident hope in the fulfillment of daily, reasonable wishes, builds an essential share in this "surplus" (basic mood). Among these are to be included even further aims and problems which we have hidden in our lives. In the "illusion" it is a question of an optimistic belief, of a firm confidence that we shall reap the success that we have aimed at in some direction or in some form. Illusion is anticipation accompanied by a pleasantly expectant emotion of gifts from the future. Its opposite takes the form of disillusion, fear of failure, apprehension and gloomy foreboding (pessimism). Rob man of his illusions and he becomes sick! Life becomes insufferable to him unless the capacity remains with him to conjure up retrospective illusion, that is, ideal memories of a time long since past. Destruction of the illusion through bitter reality is disappointment: unfulfilled wishes, or those fulfilled in the opposite sense, especially such in the sexual sphere or in that of personal success (violation of honor, of vanity), put our emotional attitude to a severe test.

Destruction of illusion, wishes, desires, especially in the sexual sphere, or discharge of such in a way contrary to the primary instincts, or cultivation of a forbidden secret "twilight life," with thereby an inevitable release of a secret passion with consequent disappointments (for instance, some passive or active sexual relations side by side, with their sweet but only briefly enduring secrecy, with their deceptions and later shame, and so forth)—all these lead, as shown by experience, especially in natures of deeply ethical or religious trend, to deep, long continuing collisions and to conditions of depression. Every single painful experience in this sphere (every "act" in such a drama) can eventually be of vital significance for the future life, for the wider development and fate of the individual; at any rate, every single insult to our instinctive life endures to a considerable extent, even if it is conquered provisionally, in the latent state; the conflict still glows under the ashes, and can become the point of origin for new collisions, whenever a new, acutely serious experience of related form appears. In the brain tissue the

"complex" exists latently to a great extent, together with the stimuli of a visceral nature once accompanying the passionate outbreak.

The visceral phenomena can break out intermittently either of themselves or in connection with the ideas connected with the experience, and also with subjective causality taking false directions (removal to remote moments unconnected with the phenomena), and can prepare new suffering and new anxieties for the individual to whom the true connection between his somatic troubles and the experience is hidden, and who frequently cannot longer remember spontaneously the details of the experience ("defensive amnesia"). Freud has made many important observations in this sphere. Bleuler and his pupils have studied these phenomena further, and have termed the slumbering residue of such experiences, emotionally toned complexes. If a suitable "reaction" does not take place, then, according to Freud, a "conversion" into somatic disorders takes place. These observations have led to, in their broader application, the theory, opposed on various grounds, of "psychoanalysis" with its complicated symbolism (Jung, Ricklin), which is known to us all. Veraguth has pointed out to us that "complexes" of various types and sources, among others, through the so-called "psychogalvanic phenomena," can be brought objectively to demonstration, a fact the authority of which one cannot doubt. All these things gave rise years ago to much discussion in our society!

Unsolved, affectively toned complexes, leading back to a series of definite experiences, especially those of a sexual nature, which slumber in the depths of the soul, in individuals (generally, indeed, "degenerates") disposed thereto (especially the feminine sex), often lead to *phobia*, to hysteria, or else finally to "neurasthenia," as shown by experience. This fact I can confirm without further discussion.

The connection between the moments just now mentioned I would like to clarify biologically. Three things are to be noted in the psychopathology of such conditions:

1. The moment of forgetting. Of course it is not a question of a total exclusion of the disturbing memory from consciousness, but only of the incapacity of the patient to think of it spontaneously, that is, on inquiry, to remember it, although great psychic injury had once produced it in him (see Bleuler, Freud, Frank, Jung, and others). Such "forgetting," in my opinion, might be considered biologically, perhaps, as a means of defense against offensive perceptions and things. One wishes that he had not lived through it; one

might thrust them away—ignore them. This defensive apparatus functions often automatically and very promptly; even under physiological conditions it comes into activity before the individual has formed the resolution to remain ignorant! The central nervous system eliminates the most harmful factors, and there follows an inhibition of the spontaneously associative ekphorie. The provisional defense has taken place.

The question now follows: Is the whole harmful phenomenon done away with, thrust back, made harmless through forgetting, even in the visceral, or the sympathetic system, even to the inner secretions (which, however, had already produced their chemical affects)?

No, there often occurs only a "slackening" as respects the cortical structures in question, that is, between anatomical basis of the thought processes and the elements (nerve cells, fibrillar bundles, molecular substance?) governing the inner secretions (innervation of the endocrine glands through mnemic stimuli). These last, once "flooded" or "overladen" ("affectively toned experience") can become a latent, that is, mnemic source of stimulation, and many a "psychic" ("logically" connected) form of excitement can be brought under that jurisdiction. In the latent state such forms of excitement might now allow a relatively independent existence, less comfortable to the bearer of it. They are generally labile, that is, always ready, to break forth from their concealment, perhaps in a suitable allusion (word, symbol), and in violent manner to exert their influence in various peripheral, visceral innervation spheres: the patient suddenly blushes or bursts into tears; his heart palpitates, and whenever the affect-laden, slumbering "complex" is touched in any way. Often the eruption is clearly "fermentative," as in the burning out of a powder fuse. This visceral function can be connected with thyroid glands (vagus system), to the adrenals and the chromaffine bodies (sympathetic system), but also to the sexual glands, the hypophysis, epiphysis, which are connected directly with the brain substance through the lymph spaces and fibrillar bands (Edinger). The related secretions bathe, indeed, all parts of the cortex and even deeper lying parts of the nervous system (through mediation of the blood or of the cerebrospinal fluid; after filtration in the choroid plexus?), whereby in an affective outbreak various even somatic phenomena can be brought to light: glycosuria, heightened bile secretion, heightened heart action, pupil dilatation, blushing, exophthalmos, diarrhoea, vomiting, localized pains, and so on (attacks). Anxiety is above all the initial symptom (reactive

excitement as to some danger believed to be imminent). The anxiety attack bears unmistakable signs of a true intoxication (products of inner secretion still unknown to us). Biologically the anxiety presents a suddenly aroused conflict to preserve the immediate existence (in the idea of the patient attacked). It is characteristic here that the activities which physiologically are most important for the patient seem directly attacked, even if only in the delusion of the patient: heart action and respiration. The emotion that the heart is standing still forces its way in, a stroke or some other evil or catastrophe is imminent, and so on, recedes from consciousness; the patient feels an intolerable load on his breast, cannot breathe; pallor, bursting out of perspiration, a jumping pulse, set in; the pupils expand; exhaustion ensues. The patient feels exhausted, irretrievably lost, and struggles for the preservation of life for that very moment, though in reality his life is in no danger. Restless torment seizes the patient; he longs passionately for release in any form, only not through death. A few minutes, and all is past, but exhaustion and intense fear of a return of the attack remain. We know that such conditions are but the daily bread of neurologists.

(To be continued)

SOCIETY PROCEEDINGS

CENTRAL NEUROPSYCHIATRIC ASSOCIATION

SECOND ANNUAL CONVENTION OF THE CENTRAL NEUROPSYCHIATRIC ASSOCIATION, HELD AT ST. LOUIS, OCTOBER 20, 1923

On October 20, 1923, the second annual convention of the Central Neuropsychiatric Association was held in St. Louis, Missouri.

The morning session, held at Barnes Hospital, began with a neurological clinic by Dr. Ernest Sachs and assistants.

Dr. Greenfield Sluder, rhinologist, read by invitation, a paper on Sphenopalatine Neuralgia and Lower Half Headaches. Dr. Sidney Schwab and assistants (Dr. A. B. Jones, Dr. A. D. Carr, Dr. G. B. Smith), presented eight cases in a general neurological clinic, consisting of: (1) Glioma, frontal lobe; (2) Tic or cortical fits following mumps in a man aged seventy-two; (3) Suprasellar tumor; (4) Left cerebellar lesion, invading meninges; (5) Paretic seizures with respiratory failure; (6) Muscular atrophy with subjective sensory symptoms; (7) Mental depression with a series of conscious conflicts and an inferiority complex; and, (8) Case showing the submergence of psychical symptoms by minor complaints and minor findings.

A clinic of Neurosyphilis in Children was presented by Dr. P. C. Jeans, by invitation.

The afternoon session, which was held in St. Johns Hospital, was opened by an Endocrine Clinic presented by Dr. Wm. Engelbach, followed by an illustrated discussion of pituitary disease.

Psychiatric papers were then presented as follows:

Mental Conditions Associated with Deafness, Dr. L. B. Alford.
The Psychiatric Factor in Vocation Incapacity, Dr. F. M. Barnes.

The Development of a Mental Hygiene Program in St. Louis, Dr. M. A. Bliss.

Report of a Family of Four Generations Presenting Sixteen Cases of the Hypertrophic Neuritic Type of Progressive Muscular Atrophy, Dr. M. L. Perry (Topeka).

The annual dinner was held at the University Club, at which retiring president, Dr. Arthur S. Hamilton, of Minneapolis, made the presidential address dealing with available facilities for special training in neuropsychiatry, and Dr. Hugh T. Patrick, of Chicago, gave a survey of European psychiatry as observed in his recent trip abroad.

Retiring officers are as follows:

President, Dr. Arthur S. Hamilton, Minneapolis.

Vice-President Dr. Lawson G. Lowrey, Minneapolis.

Secretary-Treasurer, Dr. Karl A. Menninger, Topeka.

Counsellor, Dr. Peter Bassoe, Chicago.

The officers elected for the succeeding year are:

President, Dr. George A. Moleen, Denver.

Vice-President, Dr. Herman M. Adler, Chicago.

Secretary-Treasurer, Dr. Karl A. Menninger, Topeka.

Counsellor, Dr. Arthur S. Hamilton, Minneapolis.

Twenty-six neurologists and psychiatrists of the central and western parts of the United States were voted into membership.

Number of members present, 40; total attendance, 85.

The Central Neuropsychiatric Association was formed with the idea of affording better mutual acquaintanceship among the neuropsychiatrists of the central and western states and provinces. Annual meetings are held at which the men in the convention city demonstrate their clinical and research activities and facilities. The program committee is composed of these local members and secretary.

KARL A. MENNINGER, SECRETARY, TOPEKA

CURRENT LITERATURE

I. VEGETATIVE NEUROLOGY.

2. ENDOCRINOPATHIES

Fitz, R. HYPERTHYROIDISM AND DIABETES MELLITUS. [Arch. Int. Med., March 1921.]

In this clinical study the outstanding features of 39 hitherto unpublished cases of diabetes complicated by thyroid disease are reported. He believes that the diabetes usually follows the thyroid disturbance and has a tendency to run parallel in severity with the thyroid intoxication. Cases of toxic thyroid disease with diabetes improve considerably if the hyperthyroidism is relieved.

Terrien, E. OCULAR SIGNS OF DIABETES. [Paris Médical, October 1921, XI, No. 43.]

This short clinical paper calls attention to certain ocular disturbances such as disturbances in accommodation and refraction in diabetics. He ascribes diagnostic value to the mode of their onset. Thus myopia is a frequent complication of constitutional disease, especially syphilis and diabetes.

Holst, J. GLYCOSURIA AND DIABETES IN EXOPHTHALMIC GOITER. [Acta. Med. Scand., June 1921, LV, No. 3. J. A. M. A.]

Holst declares that the relationship between the thyroid gland and the pancreas explains the glycosuria sometimes noted in exophthalmic goiter. The pancreas is evidently responsible for the severer forms of glycosuria under these conditions. He reports eight cases of exophthalmic goiter with not only alimentary but spontaneous glycosuria, including three which must be considered as a combination of exophthalmic goiter and true diabetes, the symptoms becoming aggravated or attenuated in each to correspond. In nine cases of exophthalmic goiter on record the pancreas was found pathologic. From these and other data cited he draws the practical conclusion that tests for sugar should always be applied as a routine measure in cases of hyperthyroidism, and that diabetics should always be examined for hyperthyroidism symptoms. If sugar is found in a case of exophthalmic goiter, the patient should be put on a strict antidiabetes diet. On the other hand, if symptoms of hyperthyroidism are found in a diabetic, we must consider the possible benefit from reducing the size of the thyroid, thus doing away with part of the factors responsible for the clinical picture. The depressing effect of the roentgen rays on the thyroid might be utilized. The inhibiting action

on the pancreas, normally exerted by the thyroid, becomes excessive when the thyroid is functioning to excess; reducing thyroid function will release the pancreas from this pathologic influence, and reduce the requirement of calories. Diabetes thus steps into line as a surgical affection, he says, while exophthalmic goiter joins the list of metabolic disturbances amenable to diet. The details of 20 cases of combined hyperthyroidism and sugar intolerance are given, with the necropsy findings in some.

Gorke. FASTING IN TREATMENT OF DIABETES. [Arch. f. Verd.-Krank., December 1921, XXIX, No. 1-2.]

That the starvation treatment of diabetics is quite unfavorable is the general trend of the author's study. Minkowski, however, has reported that in 17 of 19 diabetics from twelve to sixty-two years of age, the urine had been freed of sugar by starvation, and in most of them acidosis was prevented. In two very severe cases the glycosuria and acetone were markedly reduced. One of these patients left the clinic and ate large quantities of fat and protein. He developed acidosis and coma, and died eight days after leaving the clinic. Diabetics weighing about 60 kg. do better when restricted to 1,500 or 2,000 calories containing only 50 or 75 gm. protein, according to Gorke's ideas. He aims to have them weigh the same when they leave as when they entered the clinic, and to keep up the restrictions at home. The tolerance is reduced if over 2,500 calories be taken.

Kylin. HYPERTONUS AND DIABETES. [Zentralbl. f. inn. Med., November 12, 1921.]

The clinical relationship between alterations in blood pressure in diabetics is here studied. Fifty-eight cases of diabetes, 42 of whom were above and 16 below forty years of age were examined, and it was found that in the juvenile form of diabetes the blood pressure was usually within the average figures. In the senile form there was a rise of blood pressure at times to 180 mm. The readings, however, varied considerably in the same individual mornings and evenings. The almost invariable existence of hypertonus in diabetes in the aged suggested to Kylin that in cases of hypertonus in which there was no glycosuria there might be nevertheless a diminished tolerance for carbohydrates, and that cases of hypertonus without glycosuria might later be transformed into diabetes mellitus. He has, indeed, observed a woman, aged forty-four, who was under treatment in 1919 for simple nephrosclerosis, and was admitted to hospital in 1920 for diabetes mellitus. He has recently begun to test the carbohydrate tolerance of cases of hypertonus, and has occasionally found a diminished tolerance for carbohydrates, though not enough for glycosuria to appear when the patient was on ordinary diet. As the tendency to a rise of blood pressure and a diminished tolerance for carbohydrates often appears to occur in advanced life, Kylin sug-

gests that the same morbid process—namely, an anomaly of the internal secretions—is the cause of both symptoms. It is well known that in certain endocrine disturbances, such as Graves's disease, myxedema, acromegaly, and, according to some authorities, in diabetes also, there is an increase of lymphocytes in the blood. Kylin has therefore made an investigation to see if the same change occurred in hypertonus also, and found that of 16 cases examined all but two showed a lymphocytosis of from 40 to 50 per cent. As the significance of lymphocytosis is not yet established, Kylin does not wish to draw any conclusions from this, but points out that the hypothesis of an endocrine anomaly being the cause of simple arterial hypertension is supported by the blood picture.

Mördre, S. K. DIABETIC COMA WITH RENAL INSUFFICIENCY. [Acta. Medico Scandinavica, November 7, 1921, LV, No. 5.]

In this clinical paper a case of fatal diabetic coma in a young woman with hyperglycemia of 0.9 per cent and ketonemia is discussed. Her blood urea was 2.57 per thousand and the glycosuria was 4 per cent. She had no ketonuria and no Gerhardt reaction.

Banting, Best, Collip, Campbell, Fletcher. PANCREATIC EXTRACTS IN TREATMENT OF DIABETES MELLITUS. [Can. Med. Assoc. Journ., March 1922, XII, No. 3.]

The day of the pancreatic hormone seems to be breaking. These authors have prepared a concentrated internal secretion of the pancreas. They have tested it out in clinical work after extensive pharmacodynamic experimentation. They conclude that: (1) Blood sugar can be reduced even to the average. (2) Glycosuria may be abolished. (3) The acetone bodies disappear from the urine. (4) The respiratory quotient shows increased utilization of carbohydrates. (5) Improvement in the general condition of these patients was observed and the patients report a subjective sense of well being and increased vigor.

Leclercq. OVERNUTRITION IN DIABETES. [Journ. of Met. Research, February 1922, I, No. 2.]

This short note indicates that fat or alcohol added to the diet in two cases of severe diabetes can induce the production of hyperglycemia.

Allen and Wishart. ALCOHOL IN DIABETIC DIET. [Journ. of Met. Research, February 1922, I, No. 2. J. A. M. A.]

The experiments on two patients with severe diabetes support the prevailing belief that ethyl alcohol is not converted into sugar in the body. At the same time, they are interpreted as signifying that the addition of calories in the form of alcohol in excess of the patient's caloric tolerance produces a return of glycosuria and other diabetic symptoms. The experiments also corroborate the prevailing view that alcohol is not converted into acetone in the body. No antiketogenic

action was demonstrable; on the contrary a slight production of acetone seemed to be caused when alcohol was given in considerable quantities. Luxur diets formed by the addition of alcohol or a mixture of fat and alcohol to a standard diet gave rise to very much less acidosis both chemically than similarly excessive diets built up by the addition of fat alone. The former therapeutic use of alcohol is thus justified, with respect to the lessened danger of acidosis when part of the fat of a high caloric diet is substituted by alcohol. The experiments do not establish such a fact for undernutrition diets, or warrant attempts to prevent combustion of body fat by administration of alcohol. On the contrary, the conversion of an undernutrition diet into a luxur diet by addition of alcohol may result in an actual increase of acetone. With additions of alcohol, just as with additions of fat, the high caloric rations which were hoped to increase the weight and strength of emaciated patients failed to do so. As usual, the patients were stronger and more comfortable on diets which controlled their diabetes than on higher allowances which produced a return of diabetic symptoms.

Allen. RELATION OF PANCREAS PATHOLOGY TO TREATMENT IN DIABETES. [Journ. of Metabolic Research, February 1922, I, No. 2. J. A. M. A.]

In this interesting pathophysiological study Allen emphasizes the fact that the pathology bears an important relation to the treatment and prognosis of diabetes. While a true chronic pancreatitis or recurrence of acute or subacute attacks must be considered in some cases, the pathologic findings give the impression that from the standpoint of primary etiology the diabetic pancreas in most cases represents a burned out conflagration, and that the chief or sole cause of aggravation of the condition lies in hydropic degeneration of islands. This interpretation alters the former conception of diabetes as an inherently progressive disease, and affords ground for regarding the average case as the consequence of damage of a vital organ, which is in danger of further injury chiefly or solely from functional overstrain. Clinical evidence to date corroborates this view in showing that by sufficiently thorough dietary control the downward progress of most cases of diabetes is either halted or almost indefinitely delayed.

Allen, F. M. THE PATHOLOGY OF DIABETES. III. NERVOUS INFLUENCES IN THE ETIOLOGY OF EXPERIMENTAL DIABETES. [J. Metabol. Research, 1922, I, No. 53.]

Allen here concludes from his studies that no influence of emotion upon the production of diabetes could be demonstrated. Complete separation of a pancreas remnant from its original nerve-supply fails to give rise to diabetes or to any demonstrable lowering of assimilation. The full endocrine potency may be maintained without stimulation or regulation from any special nervous centers. Isolation (?) also fails to affect the occurrence or rate of hydropic degeneration in the strains. This rose

parallel with the course of the diabetes the same as when the nerve-supply is left undisturbed. The experiments are not conclusive since separation from the vegetative nerve net is an impossibility.

Allen, F. M. THE PATHOLOGY OF DIABETES. IV. THE INFLUENCE OF CIRCULATORY ALTERATIONS UPON EXPERIMENTAL DIABETES. [J. Metabol. Research, 1922, I, No. 89.]

It has been possible to reduce the arterial supply or venous drainage of pancreas remnants and also to increase the arterial supply to a slight extent. None of these circulatory changes affect the assimilative function or the pancreatic structure. The experiments are supposed to demonstrate that no support for any circulatory or vasomotor theory of the etiology of diabetes can be upheld.

Kraus, W. M. INVOLVEMENT OF PERIPHERAL NEURONS IN DIABETES MELLITUS. [Arch. of Neur. and Psych., February 1922, VII, No. 2.]

Speaking from the actual pathology of the nervous system in severe cases of diabetes, Kraus shows that involvement of the motor cells and roots or of the intramedullary portions of the sensory roots and their continuance within the spinal cord can be demonstrated. The higher synaptic pathway connections in the mid-brain, pons and medulla, may be similarly affected. Both motor and sensory involvement may occur at the same time. Clinical evidence of primary extramedullary involvement of the peripheral neurons, that is, peripheral neuritis, has not been obtained by the author.

Redwood, F. H. PITUITARY HEADACHE. [Virginia Med. Monthly, April 1921, XLVIII, No. 1.]

The author follows certain clinicians who have hypothecated a pure pituitary headache syndrome. He reports the histories of 11 patients whose sole or chief complaint was headache assumed to be of pituitary causation. Positive Wassermann reaction cases, nephritis cases and reflex cases, such as pelvic disease in women, he has been able to check off. All cases with any associated disease that could possibly have a bearing on the headache are not included. Some of these patients had other complaints for instance, convulsive attacks or diabetes insipidus, but all of them had headache. His chief reasons for assuming the headache were based on the X-ray pictures of the sella turcica. Seven had very small, and four had sellas normal in size but the clinoids enclosed the fossa. Six had headache every day, three every two or three days, and two once a month. One patient had been troubled with headache since he could remember, the other patients from six months to seven years. After pituitary extract the headaches disappeared in eight, two were not benefited. In view of the marked variability in sella morphology and the great number of hysterical headaches amenable for a time to any new therapy the author's contentions are premature.

Slesinger, E. G. NONTHYROTOXIC GOITER. [Practitioner, November 1921.]

In this paper this observer points out that while malignant goiter is far from common, it is a most interesting condition in that the pathology of some of the varieties met with is as yet far from clear. Ninety per cent of the cases develop in a preëxisting goiter, and more than 60 per cent of the patients are over forty years of age. The epithelial tumors are particularly interesting, some of them being very much on the borderland between innocent and malignant growths. He tabulates these as follows: (1) Carcinoma, when it occurs in the thyroid, forms a hard, nodular tumor, very like carcinoma mammæ, both to feel and on naked-eye section. It behaves very much like carcinoma elsewhere, metastasizing by way of the lymphatics, and its sole peculiarity is a decided predisposition to form secondary deposits in a bone. (2) The so-called malignant adenoma is a curious tumor which occurs in a single nodule of growth. It is soft and cuts like marrow, and on section shows large polyhedral fields of cells, with numerous sinusoid blood spaces. The vesicles are irregular, and lined usually with one layer of cells, in close relation to the blood spaces. The growth tends rapidly to invade the veins, and the whole picture is very similar to a section of the developing organ. (3) Metastatic colloid goiter is an extremely rare condition, which is of very doubtful malignancy. Histologically the gland is indistinguishable from ordinary colloid goiter, and the sole unusual feature is its metastasizing power. It spreads by the vascular route, and is prone to form deposits in the bones. Even when these occur, however, cases have lived as long as seventeen years. While there is insufficient evidence by which this form of goiter can be satisfactorily grouped, it would appear probable that it is incorrect to include it among the malignant growths of the thyroid. The secondary deposits in this condition have been shown, in some cases, to secrete what is apparently normal thyroid secretion. (4) Parastruma is an interesting and rare tumor in which a hard nodular mass forms in the thyroid, grows rapidly, and speedily becomes adherent to surrounding structures. It forms secondary deposits, and runs a rapid course. Albert Kocher has shown that the cells of this type of tumor contain a considerable amount of glycogen, and considers that they are derived from remnants of the parathyroids. (5) Postbranchial goiter has been described as a distinct variety of tumor.

McCarrison, R. FATS IN RELATION TO GENESIS OF GOITER. [Brit. Med. Journ., February 4, 1922, I. No. 3188.]

McCarrison's fat hypothesis is here reviewed again. The composition of the food has an important bearing on goiter he maintains. Both in relation to its content of available iodine and in relation to its content of fats the foods must be estimated. The thyroid may early enlarge not only from insufficient intake of iodine but from relative insufficiency of iodine consequent on the presence of an excess of certain

edible fats or of free fatty acid (oleic) in the digestive tract; that the food must be considered in relation not only to the normal processes of digestion but in relation to abnormal processes that may arise from the introduction of bacteria into the digestive tube; and that variations in histologic types of goiter may result from variations in the composition of a goiter inducing food. They provide, too, new points of view from which to consider the genesis of all types of thyroid anomalies.

Anders and Jameson. RELATION OF ACROMEGAL TO THYROID DISEASE. [Am. Journ. of Med. Sciences, February 1922, CLXIII, No. 2.]

In two cases of acromegaly thyroid dysfunction was observed. The use of thyroid extract in small doses gave excellent results. Marked subsidence of all of the myxedematous features, but the headaches, speech and nervous irritability have also been greatly relieved. The bony skeleton remained stationary. An associated disturbance of the thyroid function in 33 per cent of cases of acromegaly are reported in about one-third of all cases. Hypothyroidism is more commonly associated with acromegaly than hyperthyroidism, and those combined cases which manifest myxedematous features are decidedly improved as the result of the use of thyroid.

Siegel. CASE OF HYPOTHYROIDISM IN INFANT. [N. Y. Med. Journ., March 15, 1922, CXV, No. 6.]

Cretinism as an heredity anomaly has been reported and Siegel here adds another case. Therapy was unavailing.

Hamilton. HEART IN HYPERTHYROIDISM. [Brit. Med. and Surg., February 16, 1922, CLXXXVI, No. 7. J. A. M. A.]

From personal examination of a large number of hearts in hyperthyroidism Hamilton is convinced that the great majority of hearts present no evidence of damage. Heart failure is not found in this class of cases—even when death occurs. Hyperthyroidism in the presence of (a) rheumatic heart disease or (b) middle age (over forty-five years) has a tendency to cause established or paroxysmal auricular fibrillation. In many cases of hyperthyroidism showing auricular fibrillation, the auricular fibrillation disappears after relief of hyperthyroidism by operative measures, while digitalized. Cases with auricular fibrillation without true signs of heart failure have stood operation well. All auricular fibrillation cases with hyperthyroidism can be improved by digitalization. It is suggested that digitalization has a favorable influence on the cure of auricular fibrillation in hyperthyroidism.

Fischer, J. F. THE ROENTGEN TREATMENT OF MORBUS BASEDOWII. [Acta Radiol., 1921, I, 179.]

In the beginning of his paper the author refers to the literature upon the subject of his paper, giving references. In all, the author has treated

490 cases: 231 of these were private cases, of which a third were somewhat doubtful, a third slight, and a third severe cases of the disease. A case to illustrate the difficulty in diagnosis is given and discussed. Out of all the cases only 11 were men, the majority of the patients were between thirty and forty-five years, and the duration of the disease for the most part from six months to five years. In discussing the cases the author states that those treated at hospital and those treated privately must be kept apart as they are in different social positions, which is of importance. Those treated in hospital usually are persons who earn their living by physical labor, and this increases the risk of contracting the disease. The method of treating private patients is given. Recurrence nearly always occurs in hospital cases, and the author states that this is due to overwork or domestic troubles. Taking the patients as a whole, four-fifths have benefited from the treatment and the remainder even if no better, have not changed for the worse: only one case died. Patients in whom tachycardia predominates over the nervous symptoms are those in whom X-ray treatment is most likely to fail.

Fourteen cases are then described with moderately full notes and in each case at least two photographs are given, one before and one after treatment. According to the author, improvement begins after the first series of irradiations: weight should be observed, and in almost all cases where the patient is deriving benefit there is an increase in weight, often occurring after a month after the beginning of the treatment; at first there may be a small loss of weight. The effect of treatment on the various symptoms and the duration of benefit is related. Complications of X-ray treatment are next dealt with, particularly whether there is any danger to the patient; the author believes that there is none if treatment be carried out by skilled hands. The cases which die are those in which the disease is severe, and in them spontaneous hyperthyroidism may occur with no treatment, as is shown by a case quoted. It is not denied that in some circumstances X-rays may have an unfavorable effect. As regards dosage, the author agrees with the vast majority that small doses are advisable in severe cases so as to avoid X-ray sickness; he quotes Nordentöft, who says that small doses would tend to cause hyperthyroidism, whereas large doses would not. The fact that the patient's skin is always very sensitive demands caution as a smaller dose than might be expected will often cause erythema. Various other possible complications are mentioned. The technique is then described.

After discussing at some length the views of various authors on the question of surgical and X-ray treatment in these cases, the author gives his own views on the X-ray side of the question and considers it far preferable, and says it should always be used unless there is a danger in putting off an operation, which he considers to be a very rare event. [Medical Science.]

Allison, Beard, and McKinley. ROENTGEN-RAY TREATMENT OF TOXIC GOITER. [Am. Journ. of Roentgenology, November 1921, VIII, No. 11. J. A. M. A.]

Of twenty-seven cases of exophthalmic goiter without complications, subjected to Roentgen-ray treatment, but not operated, twenty-four patients are well, both from the clinical and laboratory standpoint. The treatment has been complete for nearly eight months. The remaining three cases came to operation. One patient was definitely improved before operation, the other two patients were normal a few months after operation. Of six cases of postoperative hyperthyroidism, which had relapsed, one showed a definite cure. The other five showed no improvement. Of three cases of thyrotoxic adenoma none showed any response to Roentgen-ray therapy. No bad results or complications, which could be attributed to the treatment, have occurred in any case.

Beebe, S. R. THE MEDICAL TREATMENT OF HYPERTHYROIDISM. [Medical Record, January 28, 1922.]

During the last ten years the medical treatment of hyperthyroidism has been improved quite as much as the surgical methods. Many specific therapeutic agents have been advanced only to prove unsatisfactory. There is no specific for the disease and never will be because its etiology is too complex. At present those surgeons with much goiter experience recognize the surgical limitations and the necessity for long continued medical treatment following operation. In dealing with thyroid disease we have a constitutional defect played upon by a variety of unfavorable conditions, and the medical treatment is a regimen carefully planned to remove all untoward stimuli, nervous, chemical, and toxic which influence the gland's abnormal activity. The application of this principle calls for a complete study of the patient's actual condition. Experience alone enables one to estimate the relative weight of the varying factors in this régime, but the preliminary physical examination must go far beyond the mere determination that the patient has the four cardinal symptoms with an increased basal metabolism.

The writer outlines five cardinal principles which must be observed in the treatment of every case of toxic goiter: (1) Rest; physical, mental and emotional; (2) Diet; rigid, meat free diet and exclusion of all forms of stimulants, such as tea, coffee, and alcoholics; (3) Special study of the functions of the stomach and intestine to relieve intestinal stasis and toxemia; (4) Search for and elimination of all sources of focal and chronic infection; (5) The administration of iodine in small doses over long periods of time, the dose being adjusted to the conditions in each case.

In many cases other factors enter and special measures must be applied, but in no case can there be neglect of these principles if really satisfactory results are to be obtained: (1) Rest must be adjusted to the

patient's particular needs. Merely putting the patient to bed does not fulfill the requirements; it may in fact be undesirable and make the patient worse. It may be necessary to get the patient out of her immediate environment, it may mean removal from a quarrelsome, nagging family circle, it may mean emotional relief. The second and third factors are of great importance and are most commonly neglected. There can be no compromise with the diet question. Intestinal sluggishness is more commonly found than the irritative diarrhoea about which so much has been said. Focal infections are common and in most cases are overlooked. They must be corrected to remove the continuous toxin stimulation of the gland. (6) The writer recommends the administration of iodine. This is contrary to the usual practice. The dose is small, but is larger than that advised by other writers, notably Marine. In 95 per cent of the cases it is safe and desirable to give one drop of the saturated solution of sodium iodide twice daily in the earlier part of the treatment.

To subject a patient to psychotherapy and overlook badly infected tonsils and teeth, is not good practice, nor is it good sense. To put a patient to bed and administer antithyroid serum while neglecting to correct focal infections and intestinal toxemia, is equally bad practice. Medical treatment of hyperthyroidism is not so much an attempted application of the vague generalities and indefinite hypotheses of popular endocrinology as it is an adherence to fundamental principles in taking care of the sick. [Author's abstract.]

Béclère. RADIO THERAPY OF THE ENDOCRINE GLANDS. [Paris méd., February 5, 1921.]

This author says that at present only three glands of internal secretion—namely, the thyroid, thymus, and hypophysis—are suitable for radiotherapy. Radiotherapy is the best treatment for Graves' disease and more generally for all forms of hyperthyroidism. Enlargement of the thymus giving rise to permanent or paroxysmal dyspnea, cyanosis, and syncopal attacks, is better treated by this method than by thymectomy. Hypopituitarism, which is manifested by Fröhlich's adiposogenital syndrome, requires pituitary opotherapy, whereas hyperpituitarism and pituitary tumors, in the absence of syphilis, is an indication for radiotherapy. Treatment of arterial hypertension by radiotherapy of the supranals, as suggested by Zimmern and Cottenot, does not appear to justify the employment of the method in practice.

Nordentöft, S. CASES OF GRAVES' DISEASE. [Hosp.-tid., 1921, LXIV, 31.]

Nordentöft's advocacy of the Roentgen treatment of Graves' disease is wholehearted; of 150 cases thus treated, the first dating back five years, he has not lost one, not one has become worse, and hardly any have remained stationary. Nearly all were cured or on the way to

being so. Referring to three Roentgen fatalities associated with Graves' disease and published in the Danish medical press, he expressed doubts as to death being due to, or even hastened by, the X-rays. With regard to technique, he prefers large exposures at long intervals to frequent small exposures. On no pretext should the thymus be excluded from the area under treatment. [Medical Science.]

Nordentöft, S. FIFTY CASES OF GRAVES' DISEASE TREATED BY X-RAYS. [Ugesk. f. Laeger., 1918, LXXX, 1331-71; Zentralbl. f. Chir., 1919, XLVI, 230. Med. Sc.]

Reports of 50 cases are given. The result was very good and often very striking after one seance. The 50 cases received in all 99 irradiations on the neck, and 84 over the region of the thymus. Operations were not performed without previous examination of the X-ray treatment.

Roth. AFTER ROENTGEN EXPOSURES IN EXOPHTHALMIC GOITER. [Wien. Arch. f. inn. Med., January 20, 1922, III, No. 3. J. A. M. A.]

Roth's tabulated details of the gas interchanges and metabolic findings in four cases of exophthalmic goiter, after systematic treatment with the Roentgen-rays, confirm the unmistakable benefit therefrom in recent cases. When the case is of long standing, the course long and chronic, the radiotherapy may fail to relieve. Otherwise, even when the symptoms do not show much change, the metabolic findings testify to the great transformation realized. Alimentary glycosuria could no longer be induced, in his cases, but the effect of the radiotherapy was most manifest in the respiratory gas interchanges.

Coulaud. MEDICAL TREATMENT OF EXOPHTHALMIC GOITER [Bul. Méd., November 27, 1920, XXXIV, No. 56.]

Syphilitic exophthalmic goiter is amenable to treatment. The thyroid is rich in both arsenic and iodine and according to this observer to give either of these drugs with hyperthyroidism is contraindicated. Ovarian treatment seems to relieve the thyroid of much of the extra work. Testicle extract in men has yielded some improvement but does not compare with ovarian treatment in women. Insufficiency of the ovaries is at the base of almost all of the syndromes of hyperthyroidism is his hypothesis. (And apparently his results are as good, as reported, as any others.)

Loeb, L. HETEROTRANSPLANTATION OF THYROID GLAND. [Jour. of Experimental Med., June 1, 1920. J. A. M. A.]

Loeb states that the heterotoxins injure the transplanted cells directly and make them thereby susceptible to additional injurious influences, such as lack of good vascularization, fibrous character of the stroma causing compression of acini, and direct invasion and destruction of a limited

number of acini by fibroblasts and lymphocytes. These factors lead to a constant dying of transplanted parenchyma which is not compensated for by any appreciable new formation of tissue. The primary factor in the destruction of the heterotransplant is the injurious effect of the heterotoxins. They change the metabolism of the transplanted cells which now exert an effect on the blood vessels, connective tissue cells, and lymphocytes which differs in certain respects from that exerted by normal as well as auto and homoiotransplanted cells. These secondary reactions contribute to the destruction of the heterotransplants.

Boggs, R. H. TREATMENT OF GOITER BY RADIATION. [Am. J. Roentgenol., 1919, VI, 613. Med. Sc.]

The medical, radiological, and surgical forms of treatment of goiter are reviewed, and it is pointed out that a small or moderate-sized goiter in an adolescent female is often treated by radiation with marked advantage. So far as the mode of action is concerned it is asserted that radiation causes a destruction of glandular substance and its replacement by fibrous tissue. The degree to which these changes can be brought about can be made to vary considerably. Much depends upon the proper selection of cases, and this again on recognition of the underlying pathological changes. At the Mayo Clinic they have classified goiter as hyperplastic and nonhyperplastic. Between 1909 and 1913, of 2,917 cases coming to operation, 42.8 per cent were hyperplastic and 57.2 per cent were nonhyperplastic. Of the hyperplastic 99.2 per cent were toxic, *i.e.*, toxic hyperplastic exophthalmic goiter. The indication is to treat these potential exophthalmic goitrous cases of later life in the early stage at adolescence. Clinical observations are made as to the proper method of irradiation in severe cases. The author considers that all forms of exophthalmic goiter derive benefit from X-rays or radium rays, and that some 80 per cent can be symptomatically cured. As a preoperative treatment irradiation may also be useful to diminish thyroid overactivity when the tumor is so large as to make operation dangerous. Similarly it is preferable to a second operation in cases of relapse. It is easily understood that colloid, cystic, fibrous or nodular goiters, especially if causing marked pressure without toxic symptoms, do not derive benefit from irradiation.

Hunziker and Wyss, V. PROPHYLAXIS OF GOITER. [Schweiz. med. Woch., January 19, 1922, LII, No. 3.]

In this extensive therapeutic experiment 50 per cent of the school children in Adliswil were given a tablet containing 0.5 gm. cocoa and 0.001 gm. potassium iodid for about forty weeks. This was continued until a total of 0.04 gm. of the iodid had been given. The findings in 745 children are tabulated, comparing 339 treated with 406 nontreated ones. The results confirm the author's assertion made some years ago that the thyroid in mammals hypertrophies as a defensive reaction to

iodin starvation, and it returns to average in size and function when iodine is supplied. The minute amounts given these children at Adliswil answered the physiologic demand; more would have been deleterious.

Berry, James. ON A FURTHER SERIES OF 500 GOITER OPERATIONS WITH SPECIAL REFERENCE TO AFTER-RESULTS. [Proc. Roy. Soc. Med., 1921, XIV (Surg. Sect.), 89; also Brit. J. Surg., 1920-21, VIII, 413. Med. Sc.]

Berry devoted this paper to a careful review and analysis of his cases, and included all cases in which he had removed any portion of the thyroid gland during the period from January 1, 1913, to December 31, 1919. The cases were 500 in number and formed a direct continuation of a previous series. His analysis of the sex and age incidence showed only about 12.5 per cent of the patients were males, and the age at which the disease was most common was between thirty and fifty. Five girls, however, were aged only fourteen. These were all examples of parenchymatous goiters which were causing dyspnea and failing to respond to medical treatment. Operations in children below the age of puberty were hardly ever necessary and should never be undertaken except for some specific reason. Among the male patients, seven were over seventy years of age and all made good recoveries.

In considering the pathology of the lesion he found that 198 cases were encapsuled lesions, adenoma, or cysts, and 302 were nonencapsuled. These included 13 examples of malignant disease. He states that pathologically the above classification is not strictly accurate but is convenient clinically. He lays stress upon the inefficiency of modern classifications, but points out that the terms in use are of clinical value. The series includes only three cases of inflammation. There were, however, 79 operations for exophthalmic goiter, and he points out that these cases should be sharply distinguished from false exophthalmic goiters, that is, localized lesions which may give rise to tachycardia and tremor but never show any exophthalmos. Of the malignant cases some were of the papilliferous variety, one was an endothelioma, and two were sarcomata.

In this group the first sign for operative interference was dyspnea, 74 operations being undertaken for this cause. Discomfort and deformity or hyperthyroidism were the next most frequent indications. In discussing the question of dyspnea he points out that when long persistent it may lead to other complications, such as dilatation of the heart, tachycardia, and chronic lung trouble. In no case had he to perform a tracheotomy, as he holds very strongly that the removal of the source of the pressure, namely, the goiter, is a far more satisfactory procedure. He points out, however, that after the goiter has been removed there is sometimes a danger of tracheal collapse. When the trachea is bilaterally compressed the removal of one lobe may be quite insufficient to relieve the dyspnea or indeed may make it worse, as this may lead to kinking,

which usually causes fatal suffocation. He also points out that a unilateral enlargement of the thyroid may be considered the cause of the dyspnea, whereas the opposite lobe, which may be the real offender, may lie deep behind the sternum within the thorax. He lays stress upon the difficulties of diagnosis in doubtful malignancies and states that a positive diagnosis of malignancy is almost impossible, although there may be points which give rise to suspicion. It is important, in making a diagnosis, to determine whether the thyroid moves upon the larynx or the trachea, for in the malignant cases it is likely to be fixed. He gives a full review of the operations which have been undertaken for the different varieties of disease and also a full description of his method of removing an intrathoracic goiter. In nearly all cases drainage was employed and in only seven of the 500 cases was it omitted. The mortality was extremely low, for there were only two deaths in the simple goiters, three in the exophthalmic goiters, and two in the malignant diseases. Of the 500 cases he had traced all but 14 cases. Of the total 342 of the simple goiters, 53 of the exophthalmic goiters and five of the malignant cases were quite well.

Sekiguchi, S., and Ohara, M. RESULTS OF OPERATIVE TREATMENT OF EXOPHTHALMIC GOITER. [Tohoku Jl. Exper. Med., Sendai, July 17, 1920.]

The authors report blood findings in ten cases before and after operative treatment, and after medical and Roentgen-ray treatment in five others. They find that the number of polymorphonuclear leukocytes increases while that of the lymphocytes decreases. This is also observed after other operations on the neck, and is usually transient. The blood pressure declines, usually more after thyroidectomy than after ligation of the vessels after operative treatment. The symptoms generally disappeared more or less completely and the impaired nutrition improved.

Pemberton. SURGICAL MANAGEMENT OF TOXIC GOITERS. [Brit. Med. and Surg. Journ., February 23, 1922, CLXXXVI, No. 8.]

In the period between July 1, 1920, and July 1, 1921, 1,954 patients with various types of goiter were operated on in the Mayo Clinic. The author reports some of the results: One hundred and one had ligations only. Eighteen hundred and fifty-three patients had partial thyroidectomies. In 465 the thyroidectomy had been preceded by one or more ligations. Thirty-five patients died, a mortality of 1.78 per cent. Eight of the 996 patients with simple goiter, unassociated with hyperthyroidism, died, a mortality of 0.8 per cent. Four of the 281 patients with hyperfunctioning adenomatous goiter died, a mortality of 1.4 per cent. Twenty-three of 677 patients with exophthalmic goiter, on whom 1,224 operations were performed (ligations and thyroidectomies), died, a mortality of 1.87 per cent, by operations, and 3.39 per cent by patients. Fourteen of the patients (2.39 per cent) were of the 585 who had a

thyroidectomy, and nine (1.4 per cent) were of the 639 who had ligations. Five (22 per cent) of the patients who died had recurrent goiter; they represent 8.9 per cent of the 56 patients in whom the disease recurred. Death seemed to result chiefly from the following: (1) accident, three; (2) intense hyperthyroidism, seven; and (3) moderate hyperthyroidism, plus pulmonary complications due to the patient's lowered resistance incident to the long continued progress of the disease or to some intercurrent cause, such as hemorrhage or infection, twelve. In one patient the operation failed to check the progress of the disease. Deaths are preventable to a large extent in severe hyperthyroidisms he maintains. Either an error is made in selecting the patient for operation or in selecting the operation for the patient.

Lobenhoffer. LIGATION OF ARTERIES IN GOITER OPERATIONS. [Zeit. f. Chir., October 23, 1920, XLVII, No. 43.]

This author is an advocate of ligation of all four arteries. Five hundred operations have been done in this manner by him. Tetany directly following the operation occurred only once. Parathyroid tablets brought improvement. In another patient all of the patient's hair came out, but later grew in again. These were his only accidents. Upon recent reëxamination of fifty of his patients that were operated on over a year previously, two of them, though they felt entirely well, presented a mild disturbance of the facial nerve and two had suffered from temporary falling out of the hair.

Comby, J. A CURABLE PARATHYROID INSUFFICIENCY, IDIOCY. [Archives de Méd. des Enfants, May 1921, XXIV, 303.]

Comby describes a new form of curable idiocy, due to parathyroidal insufficiency: it is nonmyxedematous, and there are no pseudolipomatous masses, no large belly, no scanty, harsh hairs, nor the special facies of sporadic cretinism. It is not congenital, but is acquired during the first months or years of life. In addition to mental debility, there are motor jerks, fibrillary tremors, and convulsions, etc. But there is no arrest of growth such as occurs in myxedema. This form of idiocy yields to parathyroid opotherapy even more quickly than myxedema to thyroid treatment. [Leonard J. Kidd, London, England.]

II. SENSORI-MOTOR NEUROLOGY.

4. PONS, MEDULLA, MIDBRAIN, EPIDEMIC ENCEPHALITIS.

Houda, E. O. MYELOENCEPHALITIS. [Northwest Medicine, July 1921, XX, No. 7. J. A. M. A.]

Houda describes finding a "virus" which showed as a very small active "micrococcus" in twenty-eight cases. The blood was taken from veins, transferred to sterile tubes, the ends of which are hermetically sealed in a gas flame to prevent contamination. After incubating for seventy-two hours or more, a vital slide made from the serum shows the virus more readily than a fresh blood slide. Seemingly the multiplication of this virus reaches a certain degree, after which further growth ceases in the same tube of blood. Transplantation has failed to produce a luxuriant growth on many culture mediums, aerobically and anaerobically. On the ordinary mediums, while no growth is visible, the vital slide shows the virus as still present after many days, if the tube is sealed to prevent evaporation and drying. In only one case was a growth obtained of what appeared as the same virus, but considering the method there is room for doubt. In one case a section of the cord was obtained postmortem aseptically. The culture medium used was a fresh calf brain. This was sectioned technically; a streak inoculation produced a growth in thirty-six hours along the line of inoculation. A vital slide on this was identical in appearance with the virus as seen in the fresh specimen, a very small active micrococcus. In six cases there were positive cultures on ordinary mediums. These showed no parallel characteristics. Each culture had peculiarities of its own. Morphologically they were classified as cocci and diphtheroids. In the blood tubes these types were readily differentiated from the small micrococcus present in practically every encephalitis case.

Watson, G. A. ENCEPHALITIS LETHARGICA INVOLVING CHIEFLY THE CEREBRAL CORTEX. [Neurol. and Psychopath., 1920, I, 1. Med. Sc.]

The patient, a woman aged twenty-eight, presented at first a picture of agitated melancholia and remained in this condition without change for four months. Then for three days she seemed to be very ill and vomited several times. During the ensuing two months she improved mentally, but began to complain of pain in the back and down the left thigh, and she limped when walking. This condition continued for another two months, when she lost her power of speech and the right side of her tongue became paralyzed. She was drowsy and lethargic. Eleven days later complete right hemiplegia with anesthesia and analgesia was observed. Optic neuritis of moderate degree was present. A week later the hemiplegic limbs showed a marked muscular atrophy and she died the following day. During her illness her temperature was subnormal and the blood count showed a definite leucocytosis. At the

post mortem examination a large area of recent softening with adherent membranes was found in the left hemisphere. After hardening, sectioning of the area disclosed streaks of vascular congestion which were not readily apparent in the rest of the brain, but no large hemorrhages. Microscopically, the meninges showed increase of blood-vessels with marked congestion and small hemorrhages. In the area of softening there was a very conspicuous increase of vessels with cellular proliferation mainly around the vessels, but in the most affected parts spreading widely into the surrounding tissue. "The proliferative elements were lymphocytoid, plasma and pseudoplasma cells, but mainly polyblasts, epithelioid and reticulate types of cells, obviously originating from the adventitia." Neuroglia cells were somewhat increased and the endothelium of the vessels showed proliferation. Small hemorrhages were found around the vessels, though they were not numerous except in the putamen and left precentral convolution, where they were rather large. Many veins showed partial occlusion by thrombi. Rarefied sieve-like spaces were noticed, especially in the white matter, containing amorphous deposits and proliferated cells. Nerve-cells were affected in the softened area corresponding to the patchy intensity of softening. The precentral and subfrontal regions, the insula and part of the temporal lobe were mostly involved. The putamen seemed to have been most affected while the remaining portions of the basal ganglia were little involved. In the temporal region the process appeared to be of longer standing and not as acute. Changes similar in character but much less intense in degree were observed in other parts of the brain, in the pons, spinal cord, and medulla, where round cell infiltrations were most marked. The nerve-roots and spinal ganglia also showed congestion, cellular proliferations, and small hemorrhages. The condition is termed a meningoencephalomyelitis on account of its widespread distribution. However, one feels somewhat doubtful whether cases of the sort are due to the same etiological factor as the ordinary types of lethargic encephalitis. [C. d. Fano.]

Hunt, J. R. TYPES OF ENCEPHALITIS. [Am. Jour. of Med. Sciences, October 1921, CLXII, No. 4, J. A. M. A.]

The results of Hunt's investigations may be summarized as follows: The large basal ganglia and especially the corpora striata, are frequently affected in epidemic encephalitis. Involvement of the corpus striatum produces three clinical types of syndromes: (1) a paleostriatal or pallidal syndrome—the paralysis agitans type; (2) a neostriatal syndrome—the choreiform type, and (3) a mixed striatal syndrome—the combined paralysis agitans-choreiform types. These three types are believed to be dependent on the existence of two distinct cellular systems within the corpus striatum. One, the pallidal system, which originates in the motor cells of the corpus striatum and links this structure with important nuclei of the hypothalamic region, and controls the various

motor activities of the extrapyramidal tracts. When this system is involved the symptoms of paralysis agitans develop, *i.e.*, paralysis of automatic associated movements with hypertonicity of the muscles and rhythmical tremor. The other, the striopallidal or neostriatal system, which exercises a coördinating and inhibitory influence on the purely motor functions of the corpus striatum. When this system is involved, chorea or spontaneous movements of the automatic associated type develop. The mixed striatal types result from involvement of both systems with the production of symptoms characteristic of each. The recognition of these two systems and fundamental syndromes of the striatum serve to explain and reconcile many peculiarities of striatal symptomatology. The pallidal or paralysis agitans type of lethargic encephalitis may be general, hemilateral or segmental in distribution. Aortive, relapsing and progressive types are also recognized. The neostriatal or choreiform type may also be general, hemilateral or local in distribution. There is an acute choreiform type; a choreoathetosis and athetoid and rhythmical types. Thalamic symptoms also occur in encephalitis lethargica. When present they consist of severe and persistent pain, with disturbances of superficial sensibility, more especially of the pain and temperature sense. Evidences of the complete thalamic syndrome are rarely if ever encountered.

Bastai, P. ETIOLOGY OF EPIDEMIC ENCEPHALITIS. [Arch. per le sc. med., 1921, XLIV, No. 212. Med. Sc.]

The present paper is in part an enlarged repetition of another one to which reference is made above. But some interesting considerations and a description of the results obtained by the intracerebral inoculations into two monkeys of 1/10th of a loopful of a 48-hours' pure culture in serum agar of the 1920 virus are new. One of the two animals did not show any special symptoms, and the experiment was considered as negative. The other, a macacus, showed phenomena of general depression with slight fever and a sort of stupor which reached its maximum at the seventh day from the inoculation. During the following days a certain tendency to recovery was noticed, so that it was considered advisable to kill the animal ten days after the beginning of the experiment. From the brain and blood (collected from the heart) the same virus was reobtained in pure cultures. The histological examination of the brain showed the presence of perivascular infiltrations chiefly due to lymphocytes and diffused to the hemispheres and basal grey formations.

According to Bastai this 1920 virus is similar to, but not identical with, that of Levaditi and Harvier, not only because it is obtainable in pure cultures and is morphologically recognizable as a very minute coccus, but also because experiments of cross-immunity, carried out by Bastai himself in Levaditi's laboratory, were attended by negative results. In addition, Levaditi and Harvier's virus is inactivated by

bile, while that of the author resists such a treatment even when prolonged for 24 hours. And the action of this virus appears peculiar as regards transmission to rabbits by means of corneal scarifications. If a loopful of a pure culture in ascitic fluid is inserted in the scarified cornea of a rabbit, it only produces a slight local reaction. But the same quantity of the same material kills a rabbit within the first 24 hours when inoculated subdurally, and a fragment from the brain of this rabbit is fatal for a third one when smeared on its scarified cornea. The same happens if a fragment from the brain of the third rabbit is smeared on the scarified cornea of a fourth one; and so on indefinitely. Strangest of all appears to be the fact that in the nervous system of all these rabbits no macroscopic or microscopic lesions were seen. [C. d. Fano.]

Da Fano, C. (3) ON ENCEPHALITIS LETHARGICA. [Brit. M. J., 1921, II, 652.]

In a previous communication on the same subject, published in collaboration with H. Ingleby (*Proc. Roy. Soc. Med.*, 1919, XII [Path. Sect.], 42), mention was made by the author of a granular pigment-like material occurring in the nerve-cells of regions of the central nervous system, where brown or black pigment is not usually found. It was also pointed out that in certain nerve-cells small, round, or oval bodies had been detected, in the middle of which a granule could be seen. The optical impression of such an appearance, when present in Bielschowsky or Nissl preparations, was that of a sort of halo surrounding each granule. When that note was written no more detailed description of such pictures was given, because it was only meant to be a short comment on a demonstration of preparations, and because the author was still uncertain as to the significance of such appearances. It was only later on that the careful examination of a great number of specimens convinced him that he had under observation something not, as far as he knew, hitherto described either in epidemic encephalitis or in other diseases of the central nervous system. As, however, the doubt of an optical illusion could not be entirely dispelled, no further communication was made on the subject, and he waited for an opportunity of some support for his conviction. This presented itself under the form of a few preparations which he was able to obtain in Paris through the kindness of H. Durand and C. Trétiakoff. The facts observed are illustrated by seven drawings and summarized in the following conclusion: in the nervous tissue from cases of lethargic encephalitis within and without the nerve-cells, minute bodies occur, to all appearance, consisting of a central basophil particle and of a delicate slightly stainable body, irregularly round or oval in shape. For these forms the term "minute bodies" is suggested *pro tempore*. The bodies are generally discrete, and provided with one granule, but dumbbell-shaped forms occur as well as others with two central particles arranged

in pairs. A relation, as yet indefinable, seems to exist between these forms and a pigment-like material occurring within the nerve-cells in places where brown pigment is not generally found. Minute forms, similar in shape, structure, and staining properties to those observed in the nervous tissue, have been traced within and without the cells infiltrating a salivary gland from an acute case of the disease. After discussing the supposition that such findings might be the product of an optical illusion associated, perhaps, with degenerative changes caused by the malady, the suggestion is tentatively put forward that they may be due to the presence in the tissues of a living agent, the cause of the disease.

In his second note the author briefly describes the method used for the demonstration of his "minute bodies." This consists of straining sections, from material fixed in alcohol and embedded in celloidin, either with 1 per cent aqueous solution of toluidine blue or with Unna's polychrome methylene blue as by Nissl's method. There is only this difference, that the sections, freed whenever possible from all celloidin, are stained for a second and third time after having been each time completely differentiated with 96 per cent alcohol. This repetition of staining and differentiating, particularly when carried out with relatively old solutions of toluidine blue, appears to have a double advantage. In the first instance, it greatly helps in imparting to the cytoplasm of nerve-cells, neuroglia cells, and other elements of the preparations, a more or less intense purple color, on which differently stained parts, such as nuclei and granules, stand out sharply and definitely. In the second place it seems to render the specimens more resistant to the injurious influence of time. In sections from acute cases of lethargic encephalitis treated in the manner described, the central particle of the supposed bodies is stained deep blue, the surrounding halo whitish-blue, while pigment granules appear colored in various shades of green.

The third note is a contribution to a discussion in the sections of Medicine and Pathology and Bacteriology at the 1921 meeting of the British Medical Association. The author cautiously comments on his findings, and gives a brief preliminary description of an acute case of encephalitis in which the disease lasted less than three days. There was intense perivascular and interstitial infiltration of all parts of the central nervous system, but especially of the white matter of cerebral cortex and optic thalamus. In contrast to what is generally seen in encephalitis lethargica, the infiltration consisted of polymorphonuclear leucocytes and macrophages, with few lymphocytes and no plasma cells. [Author's abstract.]

Whiteman, R. T., and Wilkinson, E. A. OUTBREAK OF BOTULISM AT CAMBRIDGE, IDAHO. [Jl. Am. M. A., April 29, 1922.]

The cases reported by these authors occurred among a group of persons who dined together. Of nine partaking of the same meal, eight

became ill with botulism. Six of these eight died. Only one of the nine showed no symptoms. While the symptoms in every case were practically identical, for the most part, two types of symptoms were noted, one in which the vomiting was marked and the duration of illness shorter, and one in which prostration appeared to be the predominating symptom. For brevity, only two of the most typical cases will be discussed. Blepharoptosis, paresthesias in the extremities, weakness of arm, leg and especially neck muscles, dyspnea, dysphagia and aphonia were noted in each fatal case: dilation of the pupils with sluggishness of the light reflex and Babinski's sign were present, and the knee-jerks were exaggerated: necropsies were refused by the families. Nerve stimulation, and elimination were instituted; cracked ice was given when swallowing became difficult, and physiologic sodium chlorid solution was given subcutaneously and by rectal dip. In the case of the survivors, free elimination, with magnesium sulphate, one-half ounce (15 gm.) four times daily; potassium permanganate, 1 grain (0.065 gm.); 6 ounces (200 c.c.) of water, internally four times daily, and sodium bicarbonate, 1 dram (4 gm.), internally four times daily, were used. No specimen of the suspected food or the container could be obtained for examination. Surviving members of the party stated that they ate freely of the Sunday meal and the evening lunch, excepting the canned greens, and gave as their reason for refusing them that they had a peculiar musty smell and an unpleasant taste which they were unable to describe: of seven cans of food prepared at the same time and under the same conditions as the suspected greens, five showed evidence of spoilage, though the laboratory findings were negative for *B. botulinus*.

Da Fano, C. (1) A PRELIMINARY NOTE ON THE HISTOPATHOLOGY OF EPIDEMIC (LETHARGIC) ENCEPHALITIS. [Brit. M. J., 1921, I, 153.]

Da Fano, C. (2) PREPARATIONS FROM CASES OF EPIDEMIC (LETHARGIC) ENCEPHALITIS. [J. Physiol., Proc. Physiol. Soc., 1921, LIV, 114.]

Foster, Matthias L. OCULAR SYMPTOMS OF EPIDEMIC ENCEPHALITIS. [American Journal Ophthalmology, January 1922.]

Dr. Foster calls attention to the fact that the ocular symptoms of this disease are apt to be loosely observed and poorly reported, although they are frequently the first to appear and are of great importance. Even in otherwise well worked out case reports indefinite references are often made to ptosis, diplopia, so-called strabismus, and mydriasis, without making clear the conditions of the ocular muscles or the degree to which each is involved. He says: "It seems to me that for the purpose of gaining what assistance we can from the ocular symptoms in making the diagnosis of this disease, the clinical pictures presented need to be studied with reference to the grouping of the symptoms in the individual

cases. It seems pertinent to inquire whether the successive pareses of muscles supplied by the third nerve do or do not follow any order; whether any one of these muscles is apt to suffer first, and whether any one of them is more likely to suffer a greater degree of paresis than the others. The onset of the paresis appears to be gradual in some muscles, sudden in others; the palsy of one muscle may be fleeting, while that of another, perhaps supplied by the same nerve, is comparatively stationary. Whether any constant relation between these peculiar facts can be discovered seems worth consideration. So do the relations of the pupillary reactions, and of the condition of the accommodation, to the other symptoms." Dr. Foster does not pretend to have solved any of the problems he suggests. Such a study as the one indicated can be made profitably after the symptoms presented in a great many cases have been accurately observed and published. The records of his two cases are simply a contribution to the needed data.

The difficulty of diagnosis was increased in the first case by the fact that the patient had not fully recovered from the effects of a blow on the head received some months before. He was also highly myopic, a condition in which measurement of differences of level in the fundus with the ophthalmoscope is practically impossible. When first seen by Dr. Foster he presented a partial loss of function of his left abducens, and, to a less degree, of his left levator palpebræ, both of several days' duration, and total paralysis of both inferior recti which had appeared suddenly. The pupils were quite small, reacted to light, but not to accommodation, and responded a little quicker than usual to homatropine. The right fundus was practically normal; the vessels at the margin of the left papilla bent so as to indicate either an elevation or a depression, of neither of which was there any other evidence, although with the ophthalmoscope the middle of the papilla seemed higher than the surrounding retina. No haziness of retina or papilla. These symptoms are presented in association with the general symptoms. On the following day the patient was lethargic, the ocular pareses more marked. The left eye had almost no power of movement except inward, the only muscle functioning properly in the right was the externus. The relative degrees of paresis could not be determined. Study of the reflexes suggested multiple lesions in the cortex, corpus striatum, and nuclei. Five days later the patient began to recover and the ocular pareses to improve. The last to recover was the left externus, except that paralysis of the accommodation persisted long into convalescence.

The second case was noteworthy because of the very slight general symptoms and the evanescent character of the ocular, prior to the lethargy in which the patient died. In the midst of the course of the disease recovery seemed to have taken place and the patient was discharged from hospital, only to return in ten days with the same slight symptoms. No ocular symptom lasted long, sometimes they could be observed only a few minutes. The nurse reported that an onset of

ptosis, sometimes partial, sometimes total, seemed to her a precursor of an attack of drowsiness. Among the fleeting symptoms noticed were paresis of the superior and inferior recti, total paralysis of the left internus, and inequality of the pupils. A late symptom was anesthesia of the cornea, more marked on the right. [Author's abstract.]

Cerise. OCULAR SEQUELÆ TO EPIDEMIC ENCEPHALITIS. [Méd., January 1922, III, No. 4.]

The optic nerve is rarely involved in epidemic encephalitis, but a few instances are known of scotoma and blanching of the papillæ. Cerise cites three clinicians who have reported a few cases of bilateral complete atrophy of the optic nerve. The motor disturbances may range from mere insufficiency of convergence to intense parkinsonian symptoms, and they seem to be permanent if they have survived the first acute stage. These durable disturbances from defective convergence are extremely annoying, and breed nervous and mental disorders. It is important to be on the lookout for these sequelæ of epidemic encephalitis. The patients apply for relief because near vision is becoming defective, and they need glasses. The condition is easily mistaken for defective accommodation. This may be present also, but the disturbance in vision may be cured by glasses to correct convergence, although the diplopia may still persist in lateral vision.

Hume, Nattrass and Shaw. ENCEPHALITIS LETHARGICA. [Quarterly Journal Medicine, January 1922.]

A clinical study of 22 cases. The onset was acute in 12, and insidious in 8 cases. Neuritic pains and fever in three cases were unusual initial symptoms. One of these was diagnosed sciatica. In all three the pains shifted from one side to the other, and involved chiefly the arms and legs. One case started with the symptoms of acute delusional insanity. Fever was present at the onset in all which began acutely, and probably also in those with gradual onset. The pupils showed sluggishness or failure of reaction to light in 40 per cent. Haze of vision, due to failure of accommodation, was a common complaint. Optic neuritis was not found in any case. Diplopia and ptosis were often noted. In two cases trismus was a marked feature; in one the right side of the tongue became paralyzed. Myoclonus was noted in 70 per cent, and involved chiefly the limbs, less often the abdominal and back muscles, and those of the face and jaw. The writers include, however, under the term myoclonus, cases in which jerky movements of the limbs resulted from the muscular contractions, hence perhaps the very high incidence of this sign as compared with their reports in the literature. The deep reflexes were usually normal, but in five cases there was a diminution, loss, or inequality of the knee jerks, and in one they were increased, with patellar and ankle clonus. Cerebrospinal fluid was usually negative. Post-mortem examinations in the four

fatal cases showed the usual findings, except that in one, a child of six years, numerous collections of polynuclear leucocytes were noted. An interesting feature was the presence of focal edema, both with and without cellular infiltration, which perhaps explains the fleeting character of many of the paralyses.

Levaditi, Harvier and Nicolau. AN EXPERIMENTAL STUDY OF LETHARGIC ENCEPHALITIS. [Ann. Inst. Pasteur, January 1922.]

This is a later contribution of these authors summarized in the B. M. J. Briefly, the results may be recorded as follows: The disease is caused by a virus which is able to pass through a bacterial filter and is resistant to glycerin. It is present in the nasopharyngeal secretion of the patient, as is shown by the fact that if this secretion is rubbed on to the scarified cornea of a rabbit the animal dies in a few days from encephalitis. The period of incubation in this animal is characterized by fever, by a polymorphonuclear leucocytosis, and by an increased fragility of the red cells. The virus travels from the cornea along the optic nerve to the brain. Certain anesthetics, such as chloral, ether, and chloroform, shorten the incubation period and aggravate the evolution of the disease. Pathologically, the experimental disease in the rabbit may be divided into two phases: in the first the brain shows an infiltration of polymorphs, while in the second or more chronic stage the mononuclear elements abound, either in the meninges or as cellular cuffs around the vessels. The virus is able to retain its vitality for a long time in water, milk, or in the dried condition; it is destroyed by blue and by antiseptics such as potassium permanganate; it is capable of transmitting the disease when inoculated in even very small quantities; and it does not appear to be diffusible in either glycerin or gelatin. The most marked affinity of the virus seems to be for the cornea and for the skin, and it is interesting to notice in this connection that both of these structures, together with the central nervous system, are derived embryologically from the ectoderm. So far it has not been encountered in the blood, lymphatic or salivary glands of infected animals. The relation which this virus bears to those of rabies, smallpox, and poliomyelitis is not dealt with in this memoir.

Beadles, J. N. FOUR HOURS MYOTONIC CONVULSION CAUSED BY INFLUENZA.

The case narrated below shows what a sudden severe onset may occur in influenza when the brunt of the attack falls on the meninges. A careful study of the case leaves one in no doubt that influenza, and influenza alone, was the cause of the convulsion. The effect of the shock on his wife was interesting but needs no special comment. At 3 A. M. on January 26, 1922, I was called to see F. T., a man aged thirty-eight, and found him propped up in bed unconscious.

The history was that he had never had any serious illness, but that

for the last three days he had suffered from a cold, for which he did not go to bed, nor give up in any way, as his wife was expecting to be confined. He had gone to sleep as usual the previous evening; but his wife, on waking at 2 A. M., found his face and chest wet with cold sweat, and he did not answer her when she spoke to him. She summoned help immediately from her neighbors, and as he seemed to be choking, they raised him to a sitting position in bed, which eased the breathing.

When I saw him at 3 A. M. his limbs were rigid, the arms were flexed at the elbow, and the fingers bent into the palms of the hands. The pupils were equal and slightly dilated, the eyeballs rolling upwards on raising the upper eyelid. There was slight gritting of the teeth, but the tongue was not bitten; respirations were slow but not stertorous, and the face was cyanosed; the temperature was normal.

He remained in this condition for four hours, and then the rigidity gradually passed off and consciousness returned. At 10 A. M., the only sign of meningeal irritation—supposing that to have been the pathological factor—was a certain amount of slowness in speech and perception. There was no paralysis left, and recovery was rapid and complete. Incidentally the shock caused his wife to be so deaf for a time that she did not hear unless spoken to very loudly, but this condition passed off in six hours. After the shock she received at 2 A. M., when she found her husband unconscious, she found no more signs of life in the fetus in utero, and on January 29 she was delivered of a full-grown child which had every appearance of being dead for a few days. [Author's abstract.]

Griffith, J. P. C. ACUTE CEREBELLAR ENCEPHALITIS. [Am. Jour. Med. Sciences, December 1921, CLXII, No. 6.]

This interesting summary of cerebellar involvement in encephalitis analyzes 31 cases. Four are new. In only two cases was a necropsy performed. In 25 per cent mental disturbances were present. The author has not separated the frontal lobe-cerebellar pathway disturbances from the cerebellar involvements.

Kennedy, F. BENIGN MENINGO-ENCEPHALITIS WITH PAPILLEDEMA. [Am. Arch. of Neur. and Psych., January 1922, VII, No. 1. J. A. M. A.]

Five cases are cited by Kennedy. None of them has the general coloring usually associated with epidemic encephalitis, but the fact that they have come to notice during the period of incidence of that disease and were known before that time warrants caution about asserting that the two conditions are entirely unrelated. These patients all had evidence of systemic infection, as shown by the presence of a changed blood picture, fever and general malaise. The onset was acute—in some cases sudden, with headache serving as an inadequate warning of trouble to come. In all, a period of stupor was followed by one of excitement or

disorientation, which lasted only a few days in most instances, to be followed, as a separate episode, by focal cerebral palsy-hemiplegia, hemianopia, aphasia or cranial nerve inadequacy. The rushing onset of optic neuritis late in the illness, synchronizing with amelioration of symptoms previously acquired, and its rapid amelioration in turn, are phenomena which must surely depend, for their production, on sudden blockings of intraventricular drainage by meningitic exudate, and for their disappearance, on a reconstitution of a normal fluid mechanism.

Howe, Hubert S. THE MORBID ANATOMY OF EPIDEMIC ENCEPHALITIS AS REGARDS THE ENDOCRINE SYSTEM. [Neurological Bulletin, March 1921, p. 92.]

This article is based on four cases of epidemic encephalitis studied clinically and post mortem with the object of ascertaining what changes, if any, occurred in the glands of internal secretion as a result of this disease. Some clinical manifestations indicative of endocrine disturbance were found; namely, profuse sweating, weakness, low blood pressure, menstrual disorders, impotence and metabolic disturbance resulting in increased weight. But Howe believes that these symptoms depended primarily upon lesions in the nervous system or were the result of disease toxins on the secretory organs rather than that they were due to any definite histological changes in the organs themselves. With the exception of the anterior lobe of the pituitary, there were no definite pathological alterations in the glands of internal secretion which could have resulted from the terminal illness. In the pars anterior of the pituitary a preponderance of basophilic cells and areas of focal necrosis and capillary thromboses were found. But these alterations were not prominent in any of the specimens studied, and it seemed doubtful that they were sufficient to cause disturbance in the function of the gland. The author believes that a much wider study of the internal secretory organs in epidemic encephalitis will have to be made before any definite conclusions on the subject can be reached. [Author's abstract.]

Kindberg, L., and Lelong, M. APHASIA IN EPIDEMIC ENCEPHALITIS. [Bull. d. l. Soc. Méd. des Hôp., November 4, 1921, XLV, No. 31.]

A clinical report of a case that showed aphasia for several days as the initial and only symptom. Others developed, notably a hemiplegia in the second week and the patient died.

Ducamp et al. EPIDEMIC HICCUGH. [Bull. de l'Académie de Médecine, November 8, 1921, LXXXVI, No. 36.]

A clinical report of a young woman who died eighteen days after she developed intense hiccough complicated by myoclonic movements of the legs and abdominal muscles. On autopsy, proliferation of neuroglia and degeneration of cells, mainly in the cervical spinal cord and vagospinal nucleus were found.

Houpst. EPIDEMIC HICCOUGH. [Nederl. Tijdschr. v. Geneesk., February 12, 1921.]

Houpst emphasizes the fact that none of the patients with this condition feel ill but are able to carry on their daily occupation. As regards treatment, he does not think that vertical pressure on the cricoid, which has been recommended, is of any use, but he advises pressure over the supraclavicular fossa, where one is more likely to reach the phrenic nerve. He has had considerable success from the use of a combination of the bromides in the form of Charcot's solution, a teaspoonful of which is given every two hours until the hiccough ceases. McWalter, who reported a small outbreak of hiccough in Dublin, found intranasal application of chloroform, camphor, and olive oil of much service. [J.]

Hall, A. J. ENCEPHALITIS LETHARGICA. [Lancet, 1922, I, 526.]

The writer records thirty cases of encephalitis lethargica which he has seen since October 1918. Up to then he had seen sixteen cases including the original cases recorded in the *Lancet*, 1918, I, 568.

Eleven out of these forty-six cases died (about 24 per cent). One of the cases died not from the disease itself but from epidemic influenza, six months after the onset of encephalitis, and while he was still suffering from the effects of the lesion in the midbrain. The thirty cases in this paper are divided into three groups:

(1) Mild or abortive type—five cases; (2) cases of medium severity—fourteen cases; (3) Fatal cases—eleven.

In considering the fatal cases the writer raises the question as to whether complete rest in bed is essential or desirable in the very lethargic cases, capable of getting up; especially in adults who have been previously in active work. The duration of life from the onset of definite symptoms among ten of the fatal cases was 8 days in two, 12 to 15 days in four, 21 days in three, 46 days in one. It was not possible to trace the source of infection in any of the cases. The after history of the nonfatal cases shows that on the whole most of them recovered more or less completely after a longer or shorter time. Two cases with complete paraplegia had regained their power and a third had probably only a psychic paralysis left. In two cases tuberculosis had developed, probably the lighting up of preëxisting infection, in a third fatal case, old foci at both apices were found to have become active and to be surrounded with recent miliary tubercles. No particular line of treatment was found effectual. The chief characteristic in prognosis is its uncertainty. [Author's abstract.]

Almasio. LETHARGIC ENCEPHALITIS AND INFLUENZA. [Il. Policlinico, February 14, 1921. B. M. J.]

This author states that there was an epidemic of 138 cases of lethargic encephalitis at Turin between December, 1919, and April, 1920; 4 occurred in the second half of December; 74 in January; 43 in Febru-

ary, and 17 in March; 48 died—a mortality of 34 per cent. The death rate was highest after sixty (6 deaths among 11 cases), fairly high from twenty to thirty (18 deaths among 47 cases) and from thirty to forty (7 deaths among 31 cases), and lower from ten to twenty (4 deaths among 19 cases). The mortality in children under ten was fairly high (3 deaths among 9 cases). The lowest mortality was from fifty to sixty (1 death among 6 cases). The two sexes were almost equally affected, 74 cases being in males and 64 in females; 72 had not had influenza in 1918, and only 12 had been attacked then. In 38 the disease started suddenly, and in 14 the symptoms of encephalitis appeared as a complication or sequel of influenza. All social classes were affected. No example of contagion was observed. The influenza epidemic of 1920 in Turin closely corresponded with the epidemic of lethargic encephalitis. It gradually increased in intensity from the beginning of January till the 29th, progressively diminished in February, and completely ceased at the end of March. In both influenza and lethargic encephalitis the majority of cases occurred in persons between twenty and thirty years of age. The curves of the two diseases, however, differed as regards the number of those affected in early life. Whereas in encephalitis there were very few patients between the first and fifth years of life, in influenza there were many cases at this age.

Bing and Staehelin. EPIDEMIC ENCEPHALITIS. [Schweiz. med. Woch., February 9, 1922, LII, No. 6.]

Ninety-seven cases of epidemic encephalitis are here subjected to analysis. The mortality was about 20 per cent. Suicide in Parkinsonian cases was frequent. This has a bad prognosis when of the slow tardy type. The myoclonic form proved more malignant than the choreic form. Recovery in the lethargic form in 25 per cent of the cases only. In 50 per cent Parkinsonian followed, sometimes after an interval of apparent health for several months up to more than a year. In 11 cases the tardy Parkinsonism is growing more pronounced. Of the 97 cases, only 24 of the 80 who did not die are free from sequelae.

Howell, K. M. COLLOIDAL GOLD CHLORID CURVE IN EPIDEMIC ENCEPHALITIS. [Am. Arch. of Neur. and Psych., February 1922, VII, No. 2. J. A. M. A.]

No characteristic colloidal gold chlorid curve was obtained by Howell with cerebrospinal fluids in epidemic encephalitis. When there was a color change, it occurred in the lower spinal fluid dilutions (syphilitic zone). This result agrees with those in the recent publications of Davis and Kraus, Happ and Mason, and Neal. There was no relation between the colloidal gold chlorid reaction and the duration of the disease. The colloidal gold chlorid curve apparently was not influenced by the total cell count, nor by the polymorphonuclear or mononuclear leukocytic percentages. The colloidal gold curve did not

depend on the globulin content of the fluids. There was no relationship between the colloidal gold chlorid reaction and the colloidal benzoin precipitation reaction. The Wassermann reaction with all fluids was negative. Spinal fluid from a patient with epidemic encephalitis may give a typical paretic curve with colloidal gold chlorid when there are no symptoms or history of syphilis.

Zuccola. EPIDEMIC HICCUGH. [Il Policlinco, January 24, 1921.]

Zuccola of Cuneo refers to a type of what he calls epidemic encephalitis which seems to be distinct from the prevalent form. The first cases were reported from certain parts of France, and now something of the kind is evident in Italy. Unlike the older disease, the course is mild and the inception insidious. There is slight malaise, the head feels heavy and there is a certain amount of headache and vague rheumatoid pains in the body at large. The digestive and respiratory systems are almost spared. Fever may be slight and even absent, and chilliness is more marked. There are no pareses of the ocular muscles or implication of cranial nerves. Seemingly the chief if not sole symptom is a clonus of the diaphragm, 15 to 25 to the minute, which may or may not be simple hiccough. It is not influenced by anything in the way of posture, medicines, etc. The condition is certainly not new in medical literature and the author cites many Italians who have formerly reported such cases. Hiccough has also been seen in both encephalitis lethargica and influenza, but the author believes in the autonomy of the new affection.

Reh, T. INSOMNIA FOLLOWING EPIDEMIC ENCEPHALITIS. [Revue Méd. d. l. Suisse Romande, March 1921, XLI, No. 3.]

This not infrequent syndrome is here discussed. In two cases the insomnia and agitation at night had persisted for seven and for eleven months. The children then had an attack of measles, after which the insomnia disappeared.

Fletcher. DIURNAL SOMNOLENCE AND NOCTURNAL WAKEFULNESS IN ENCEPHALITIS. [British Journal of Children's Diseases, April-June 1921, XVIII, No. 208-210.]

This author also calls attention to the wakefulness at night in this condition. Two cases are cited in which there was a remarkable degree of somnolence during the day associated with wakefulness during the night. In one case the onset was associated with pains in the limbs and diplopia.

Regan, J. C. COLLOIDAL GOLD REACTION IN THE DIAGNOSIS AND TREATMENT OF EPIDEMIC POLIOMYELITIS. [N. Y. Acad. Med., Ped. Sect., November 10, 1921.]

This paper is a study of 21 cases of poliomyelitis which were admitted to the Kingston Avenue Hospital during the autumn of 1920. A

total of 74 special fluids were examined, the fluids being taken at intervals varying from the fourth to the one hundred and twenty-third day of the disease. The curves thus obtained were classified according to the week of the disease in which the spinal fluids were taken. On this basis an average curve was constructed for each week from the first to the eighth; likewise an average curve for the fatal cases. The following conclusions were drawn from the study: There was always a reaction with colloidal gold solution in every poliomyelitis fluid examined during the acute stage of the disease. This reaction was constantly in the same zone with that of luetics. The average curves for the first and second weeks were very similar, and consisted in a gradually rising curve representing a graduated increase in reduction in ascending dilutions of the spinal fluid starting in the reddish-blue zone in 1-10 dilutions and extending into the lilac or purple zone in 1-40 to 1-80 dilutions, and then descending somewhat more abruptly into the reddish-blue zone in 1-160 and reaching the nonreducing or red zone in 1-640. In the latter weeks of the disease the average curve gradually subsided, reaching normal in a minor proportion of the cases (3 out of 10), by the eighth to the ninth week, while in the remaining 7 (70 per cent) the curve was still elevated at the ninth week. In two cases examined later than the eleventh week, one being in the fourteenth and the other in the eighteenth, the curve was still elevated in both. The earliest subsidence of the curve to normal was the twenty-eighth day. Cases presenting a persistence of the very acute curve of the first few weeks up to the ninth week or beyond commonly presented at this time either considerable residual paralysis with slight, moderate or marked polyneuritis, or else slight paralysis with a neuritis which was still pronounced. The writers were inclined to believe that there was a relationship between the duration of the positive colloidal gold curve and the acute inflammatory stage of the malady, so that when the gold chloride reaction became normal the acute period of the disease was over. If this was so the reaction should therefore be of value in determining when the rest of the acute stage might be terminated. The gold chloride test had clearly differentiated in this series of cases the various forms of meningism, tuberculous meningitis, and epidemic meningitis. The only curves with which that obtained in poliomyelitis was apt to be confused were those obtained in cerebrospinal lues and in a minor proportion of cases of epidemic encephalitis. From the former the clinical symptoms, history and Wassermann reaction would afford an absolute means of differentiation, so that no mistakes could thus arise. Most cases of epidemic encephalitis gave a paretic, mild luetic or negative reaction. It was only those which presented a mild luetic curve which would, by means of the gold chloride reaction, be indistinguishable from poliomyelitis. No close relationship was found between the cytology and chemistry of the spinal fluid and the colloidal gold reaction except in a

general way. The reactions obtained in the fatal cases although showing a tendency to produce greater reduction, did not differ sufficiently from those obtained in the nonfatal cases to make the test of value in prognosis. It must be realized that the gold chloride reaction was the most valuable laboratory test we possessed at the present time for the recognition of Heine Médin's disease. To realize its full value it must be taken into consideration with the history, physical findings, and other laboratory data in the individual case.

Roasenda. INSOMNIA AS A SEQUEL OF LETHARGIC ENCEPHALITIS. [Il Policlinico, February 7, 1921.]

Three cases, patients aged thirty-five, thirty-six, and five, respectively, after an attack of lethargic encephalitis, presented an inversion of the natural rhythm of sleep. Instead of sleeping during the night and remaining awake during the day they enjoyed a fairly good and refreshing sleep from early morning until late in the day, but were tormented by insomnia with psychomotor agitation during the night. The insomnia was not affected by any of the usual remedies, but persisted indefinitely.

Cruchet. LETHARGIC ENCEPHALITIS IN THE BORDEAUX REGION. [Gaz. hebdomadaire des Sciences Médicales de Bordeaux, January 16, 1921.]

This paper records the analysis of 145 cases which occurred between March, 1918, and October, 1920. Their classification was as follows: Meningoencephalitic form, 56 cases; pontine and bulbopontine forms, 32 cases; choreic and myoclonic forms, 15 cases; mental forms, 14 cases; epileptic form, 2 cases; hemiplegic form, 2 cases; cerebellar form, 1 case; spinal form, 6 cases; polyneuritic form, 3 cases; unclassified, 14 cases. Of the 145 cases 27 died—a mortality of 18.6 per cent, which was considerably below the figures of 35 and 50 per cent reported previously. But although recovery was frequent it was far from being always complete; convalescence was extremely slow and sequelæ persisted for a considerable time. Mentally the patients remained depressed and incapable of sustained intellectual effort for a long period, and many had to be sent to asylums. A generalized spastic condition of the type of paralysis agitans was very frequent. The prognosis of the myoclonic form was usually grave; of 12 cases, 7 died and 4 were in a desperate condition. No cases of contagion were observed. There was nothing found to justify the conclusion that there was any relationship between lethargic encephalitis and influenza or between lethargic encephalitis and acute poliomyelitis. Lethargic encephalitis was observed in both sexes, in childhood and adult life. Cases were rare after forty.

BOOK REVIEWS

Baudouin, Charles. THE POWER WITHIN US. Translated by Eden and Cedar Paul. [Dodd Mead and Company, New York.]

One commendable feature of this addition to the galaxy of the Pollyanna literature on mental therapeutics is its scholarship and charming style. It is largely a skillfully worked mosaic of Emerson and James, with frequent dashes of Coué. To the American reader conversant with the works of Emerson, Thoreau, Holmes and James it is superfluous apart from its easy flow and wide acquaintanceship with later developments of our nature philosophers.

Hobhouse, L. T. THE RATIONAL GOOD. [Henry Holt and Company, New York.]

Morals in Evolution is the title of a previous work of the author which has been reviewed in these columns. Ethical theory as a constituent part of behavior is constantly being dwelt upon by the neuro-psychiatrist, no matter from what practical angle he approaches his work. The worker in this domain sees human action arising out of ancestral impulses, and, in recent years, through the insight gained by the study of the unconscious, has commenced to pierce much of the camouflage of rationalization. Concerted rationalizations hiding behind mass psychological reactions as seen in religious and political bodies, and as fostered by open and by secret propaganda are still too firmly entrenched to be properly evaluated. The author has some inkling of this type of process going on in the evolution of ethical values and has here written an extremely simple and easy account of the general process. It is a little too general for the actual work of the clinic whether at home, in the hospital, in court or in penal institutions, but it can be read with profit.

Brown, Langdon W. THE SYMPATHETIC NERVOUS SYSTEM IN DISEASE. Second Edition. [Henry Frowde and Hodder and Stoughton, London.]

We have had occasion to commend the first edition of this small work on the sympathetic nervous system. Dr. Brown has had the opportunity of revising it somewhat so that it represents a very commendable brief presentation of many important facts about the visceral or vegetative nervous system and of some of the disturbances. It does not appear that the energetic hypothesis of man as an energy transformer is clearly grasped by the author, else his mode of dealing with this system which is so intrinsically involved in this work would have been somewhat different.

Bychowski, Gustav. METAPHYSIC UND SCHIZOPHRENIE. [S. Karger, Berlin.]

Psychoanalytic studies have given an entirely new impulse to psychiatry and as with any new thoroughgoing conceptions have tended to introduce much simplicity of interpretation which heretofore has been chaotic.

One of the most fruitful lines of research has been the study of human cultural stages as they come out of the diseased individual in thinly disguised forms. These psychological fossils, as Jung has termed them, take on a meaning of far reaching significance and give to psychopathology an importance for the understanding of the human psyche which is only just beginning to be appreciated by the older students of psychology and anthropology.

The present study is one focussed upon paleopsychology, to use Jelliffe's phrase, although entitled *Metaphysics and Schizophrenia*. He purposes to study the biological foundations of the relation of the psyche to the surrounding cosmos as it is built up phyletically in human history and as it is peeled off layerwise, as it were, in the reduction process of such a psychosis as schizophrenia.

Freud supplied the original stimulus, Jung developed the idea fruitfully and a number of other students of psychiatric problems have carried on. In recent years the principle has given interesting results, Hollos and Ferenczi have made the devolution of the personality in paresis an understandable process; Storch and the present writer have applied it to schizophrenia, Groddeck, Jelliffe and Ferenczi to organic disease of the body. This principle of the retrograde breaking down of function as a definite reversal of its evolutionary course as applied to the metaphysic of the world and its phenomena in different cultural epochs is beautifully demonstrated by this fascinating monograph.

Fumarola, Gioaechino. DIAGNOSTICA DELLE MALATTIE DEL SISTEMA NERVOSO. Parte Generale. [Casa Editrice Luigi Pozzi, Roma.]

Dr. Fumarola is attached to Mingazzini's clinic at the University of Rome, and his chief has written a short preface to this excellent volume.

In the manner of general semiology Fumarola here offers 350 pages of clear descriptive text on methods of examining for disturbances of nervous functions, with a general discussion of the significance of symptoms.

This rich collection of material is grouped in the usual manner under examinations for motility for reflexes, and for sensibility. Language, apraxia and psychical states in their neurological aspects are then dealt with; finally lumbar puncture, Wassermann tests, laryngoscopy, electrodiagnosis, radiography, are considered in their relation to neurological diagnosis.

The whole makes an excellent manual for students of neurological problems.

Metzger, John A. PRINCIPLES AND PRACTICE OF X-RAY TECHNIC FOR DIAGNOSIS. [C. V. Mosby Company, St. Louis.]

The need for the use of X-ray examinations is nowhere more imperative than in the study of the patients who come to the neuro-psychiatrist for diagnosis and treatment.

This small manual is admirably conceived and as commendably executed to fill this need. It is most excellent and deserving of more than this short encomium of its merits.

Sommer, Robert. FAMILIENFORSCHUNG UND VERERBUNGSLEHRE. Zweite Auflage. [Johann Ambrosius Barth, Leipzig.]

Professor Sommer of Giessen is well known to the psychiatric world. We have frequently had occasion to call attention to some of his excellent contributions which now number nearly 200, embracing poetry, history, social psychology, heredity, forensic psychiatry, psychopathology, neurology, psychiatry, internal medicine, philosophy and esthetics.

His contributions in the field of the present work have been some twenty in number, this volume being a complete monograph upon the general subject of genealogical research and the study of heredity, particularly of the heredity of psychical characteristics.

The first five chapters deal with the modern development of hereditary transmission factors, chiefly fashioned on Mendelian foundations.

Genealogy as a field for the study of heredity is then discussed at length. Family names, coats of arms, family charts are analyzed, as to methods of interpretation. Incest, amphimixis, and epimixis are defined and family and race described. Bodily conformation, structural unit characters and psychological factors are then taken up.

In chapter XIII the history of a family from the fourteenth to the twentieth century is presented as an example of the method of research and what it may be expected to yield, from all the stand-points previously discussed.

Pathological results of hereditary degenerative factors and criminality are then discussed and also the emergence of high types; this leads to a consideration of race hygiene or eugenics, with special emphasis given to German history, and famous German families. Finally Sommer gives an extensive analysis of his own family tree.

The book is quite unique and very interesting. It deserves a well merited place in the field of the study of heredity.

Hertzler, Arthur E. DISEASES OF THE THYROID GLAND. [Mosby Company, St. Louis.]

The author tells us this is a study which has come from work in a small country hospital. He speaks of it as a departure. For the United States this is true; for European countries, particularly the small German university towns, such studies have been emerging for many years. They have been among the most important contributions to medical science, and we welcome this new departure.

It is to be commended as well, not because of the reasons just sketched, but by reason of the fact it would be an exemplary contribution no matter where it came from. Its chief value in this respect is not really due to what he suggests, namely, that it permits an author to follow his own ideas, but that he did it at all. It takes courage perhaps more to have ideas than to present them. Conventionality is just as rampant in medicine as it is in every other activity. Medical Babbitts are all too frequent, even in high places, even though the spirit of medicine should be an efficient antidote.

As to the author's performance itself it is commendable, chiefly as a record of work. More than half of the book is devoted to this aspect of the subject. The microscopical studies are excellent.

We do not look for a philosophical presentation of the goiter problem. The author has really not shown that there could be such a point of view. He has given an excellent descriptive book about his own material and it is a welcome addition to the literature, even if not an important contribution in the sense of new knowledge.

Régis, E., et Hesnard, A. LA PSYCHOANALYSES DES NEVROSES ET DES PSYCHOSES, SES APPLICATIONS MÉDICALES ET EXTRA-MÉDICALES. Deuxieme Edition. [Felix Alcan, Paris.]

The first edition of this the most serious review of psychoanalytic doctrines in French appeared just before the war and was the last important contribution of Régis, who has since died. In this edition the junior author has borne the burden.

The general spirit may be gained from Hesnard's introduction which chiefly centers about his chief, whose eclectic spirit in psychiatry would not permit him, he writes, to neglect entirely so very definite a movement as psychoanalysis. Hesnard, however, cannot avoid waving the flag of nationalism, and thus introduces us all too early to that enemy of all real science, the setting off of one nation's science as compared to another. The "Latin" genius is offered in contrast to "German philosophy." This is but a very minor note in the introduction but one is already on the lookout for propaganda and not for real comprehension.

Happily, however, Hesnard has made the present edition a far more acceptable one than its predecessor in this respect, although all too frequently we hear rattled the old skeletons of pansexualism and mysticism—two very efficient means of raising up prejudice.

It is too much to hope that so-called scientists ever really eliminate their affective prejudices, but one looks for real insight concerning a problem when it is being critically analyzed. The present volume does not err on the affective side as did the first edition but it still shows the impossibilities of understanding that pertain to the attitudes of a looker-on rather than as one in the thick of the fray. The book displays a definite defect of first hand knowledge of actual experience although it is an excellent literary discussion. If, as the author says, he has really done some successful psychoanalyses some of his comments are absolutely foolish. Above all the authors

never really have gotten into the pragmatic attitude of seeing how an hypothesis works out by thoroughgoing application of the principle. They hem and haw on every stage in the process and show up all the objections of why a thing should not work, all actually preparatory to seeing it *at work*.

The history of every advance in every sphere of activity has shown this selfsame trend. It is to be looked for, and in many senses it is desirable that it should be so, but when one finds much misrepresentation of a situation within a criticism then such becomes annoying. We find the present volume freer from the type of actual voluntary lying about the Freudian hypotheses which is present in the majority of the works of the kind, now nearly finished their course. It also has fewer of the stupidities with which the so-called critical literature of anti-freudian ideas abounds. But it still contains too many narrow half renderings of ideas; principles are dissociated from previous postulates and made to stand as independent verities, much as a headless horseman would be called upon to function physiologically. This type of mutilation of an organizing body of pragmatically and objectively tested hypotheses is all too suggestive of the typical legal casuistry so dear to a destructive sophistry.

We should be unfair to this work were we to suggest that this is what it is. It is far from this—what we have written of is but the blemish of an otherwise very excellent and attempted fairminded presentation. One very dominant misconception is harped upon so often that we must say that fairmindedness is difficult to separate from the typical American habit of “passing the buck.” Freud is repeatedly being quoted as saying that the “sexual instinct is the only source of the psychical activity.” It matters little that for over twenty years in at least hundreds of publications this stupid misstatement has been corrected—no one can really find any such things in Freud’s ideas, except by the now familiar and odious process of taking a word or sentence or statement out of its context—nevertheless sexual prudery is still so universal that it makes a fine grandstand play against which no amount of correction will ever be availing. Thus critics keep on “passing the buck” by misinterpretation actually unmindful of the affective sources from which this scientific dishonesty arises. This book has less of this than many we know but it is not entirely free from manifest evidences of casuistry and obvious resistances. In his final paragraphs the author suggests his own defense reaction. “One can say of the psychoanalytic school that it risks practicing the culture of an obsession and of a fixed idea.”

de Saussure, Raymond. LA MÉTHODE PSYCHOANALYTIQUE.
[Librairie Payot & Co., Lausanne, Geneva, Paris.]

In these columns attention has frequently been called, not without a certain acerbity, to the many false prophets who during the past fifteen years have been asserting, with greater show of irritation and anger, that the psychoanalytic movement was an “ignis fatuis,” an obsession, a rush light, and even worse, a smudge.

Many honored names, at one time considered as ornaments to our front cover, and still held in respect for their work in the narrower fields of structural neurology, have joined the group of these dissenters. In spite of the many prophecies, cast in varied forms, ridicule, bitter animosity, blind hatred, prejudiced stupidity, the movement has gone forward, has grown, and has spread, both healthily and weedily, throughout the world. In spite of the fact that many rank and futile excrescences and appendages have lent a certain justification for the antagonisms aroused, the essential principles of psychoanalysis have had a wider and deeper reception, and have modified the entire structure of medicine, especially in neuropsychiatry.

Among the last of the "cultured" countries to openly present its support to the movement has been France. This is no place to make an effort to show the many whys for this resistance. Germanophobia is not the most important one. We believe it means that France needed psychoanalysis much less than many another nationality. Although of Latin origin, camouflage has not been a particularly essential French trait. Rousseau, Rochefoucauld, La Bruyère, Voltaire, among the earlier epigrammatists had riddled "repression" with the shafts of their brilliant wit. Voltaire's classical allusion to the English as a "nation of one hundred religions and one sauce," points the lesson of a widespread sublimation of sensuous activities in his countrymen as contrasted to the limitations of such sublimations in a folk for whom "fear of hell and the devil" held their repressions under the bondage of limited religious mass-psychological protective devices.

The present volume, although from French Switzerland, is one of a number of recent French expositions of the Freudian principles. It is one of the best.

Inasmuch as it is a simple exposé of these principles we shall not analyze it in detail since it presents little that is not already current knowledge. In one respect, however, it is noteworthy. It possesses a singular lucidity as well as good style, and is written in a most pleasing manner. It is free from controversial expressions and all in all is one of the best smaller contributions to the psycho-analytic literature extant.

Furthermore, it comes from a scion of a house whose claim to scientific preëminence for several generations has been of world-wide recognition.

de Mouchy, S. J. R. DIE ZERGLIEDERUNG DES PSYCHISCHEN KRANKHEITSBILDES BEI ARTERIOSKLEROSIS-CEREBRI. [S. Karger, Berlin.]

It is yearly becoming more and more evident that not only in such a special field as neuropsychiatry, but in all domains of medicine, that nosology as such is falling into disrepute. Classifications are group conventions. They serve as conveniences. They tend to become easily fixed and arriving there are hindrances to growth.

They become the shibboleths of the "practical" man, who, as Lord Beaconsfield has so aptly said, "is one who perpetuates the errors of his forefathers."

Kraus, in his large work on the "General Pathology of the Personality," has stated that the older nosologies are unsatisfactory. Virchow's cellular pathology, excellent as a descriptive science, is in need of revision. It needs more "why" into its "whats." A dynamic medicine is arriving, and its development is no more active than in the field of neuropsychiatry. In fact it may be stated that this newer dynamic accent first came from the camp of the neuropsychiatrist, chiefly the latter discipline of this hyphenated combination.

In the psychiatric field this newer movement has been very active, and the present contribution is one imbued with the concepts reflecting the evolution of psychiatry away from static classifications to an etiological basis of a pluralistic type.

In this study on cerebral arteriosclerosis, as seen in its psychotic manifestations, a greater emphasis is placed upon the personality foundations as giving rise to the mosaic of clinical pictures than upon the exogenous factors, so dear to the medicine of the past fifty years. Whereas a polyetiological recognition is not wanting—infections, intoxications, etc., etc., are not wiped out by any means, yet their dynamic significance is subordinate to other features in the clinical history. This study from L. Bouman's clinic in Amsterdam can be read with great profit. It is distinctly in line with Lewis' recent study on the "Constitutional Factors in Dementia Praecox,"¹ with Hollis' and Ferenczi's recent contribution to an understanding of the symptoms of paresis, and to a number of recent developments in neuropsychiatry emphasized by Kretschmer, White, Jelgersma, Specht, Jelliffe, Birnbaum, Kempf, Stoddart, and others.

It is a very readable piece of work, and indicates the trends of the newer Dutch school in psychiatry.

Stiles, Harold J. and Forrester-Brown, M. F. TREATMENT OF INJURIES OF THE PERIPHERAL SPINAL NERVES. [Henry Frowde and Hodder and Stoughton, London.]

The authors tell us this monograph is designed to place at the disposal of the general surgeon, who may be called on to deal with an occasional case of peripheral nerve injury, the experience which has been gathered from the exceptionally abundant material provided by the great war. It is hoped to map out for the surgeon who has no special experience of the subject those paths which will lead to a successful result for himself and his patient and to help him to avoid those pitfalls which have entrapped most workers at first, before they learned to look out for them.

Without much ado it may be said they have done this in a highly successful manner in a monograph of exceptional value and of exemplary character.

¹ Monograph Series, No. 35.

OBITUARY

BORIS SIDIS

Boris Sidis, Portsmouth, N. H.; Medical School of Harvard University, Boston, 1908; at one time associate psychologist and psychopathologist to the New York Pathological Institute, and the New York State hospitals; director of the Psychopathologic Hospital and psychopathologic laboratory of the New York Infirmary for Women and Children; proprietor of the Sidis Psychotherapeutic Institute; formerly associate editor of *Archives of Neurology and Psychopathology* and the *Journal of Abnormal Psychology*; author of "Psychology of Suggestion," "Philistine and Genius," "Experimental Study of Sleep," and other works; aged fifty-five; died suddenly, October 23, 1923.

DR. FORD ROBERTSON

Through the untimely death in his fifty-sixth year, of William Ford Robertson, M.D., Edinburgh has lost one whose life was wholeheartedly and devotedly given over to research. The study of pathological and bacteriological problems concerning the cause and treatment of nervous disease, insanity, and cancer had preoccupied him ever since his student days; they had filled his thoughts to the exclusion of other pursuits; and his last days were shadowed by the fear that he might not be spared to bring his studies to a conclusion. Robertson was born on the Borders in 1867; he was a student in Edinburgh from 1886 to 1891, and even during his undergraduate course he showed his bent towards pathology and his originality in technique in the work he did under Prof. William Russell, who was then pathologist to the Royal Infirmary. After graduating he held several Resident appointments, and in 1893 became pathologist to the Royal Asylum. His papers, between 1894 and 1896, on the morbid anatomy of mental disease, attracted much attention, and stimulated fresh interest in this branch of research. Their value was recognized by the creation, in 1897, of the Laboratory of the Scottish Asylums, with Robertson as its head, and to the opportunities which it afforded him he consecrated the rest of

his working days. As years wore on his interest turned more towards bacteriology as an explanation and as a means of treating organic disease. Endowed as he was with originality and vision, his hypotheses at times seemed to outrun his observations, but he was certainly one of the first workers to appreciate the importance of infections arising from the intestinal and urinary tracts, and to apply on an extensive scale the principle of immunization with vaccines. His contributions to medical literature were many. In addition to his papers on the histology of insanity, he published numerous papers on tabes and general paralysis (his researches on which were embodied in the Morison Lectures for 1906), dementia precox, and the parasitic origin of cancer. He found inspiration especially in the Italian school of psychiatry, and translated Tanzi's works into English. His last book on therapeutic immunization was published in 1891; it was the outcome of years of experience and exposition of a profound faith in this method of therapeutics. Ford Robertson was a man of tenacious convictions and boundless optimism. Never discouraged by failure, he was of the type to whom nature sooner or later reveals some of her secrets. It was, some of his friends thought, unfortunate that the condition of his work did not bring him more into daily contact with other researchers in the same field, and that he was thus deprived of the benefits of criticism and comparison of his results with the work of others. It was also his misfortune that in his early years the bacteriological school of Edinburgh was in its infancy, and that when he came to apply bacteriological methods to the problems which interested him he was of necessity self-taught. To have overcome these hindrances to the extent that he did showed him possessed of talents and industry of no mean order, and but for his premature death he would have gained much wider recognition.

Dr. Ford Robertson married in 1897, and is survived by his widow and three sons, one of whom is following his father's profession.

(Edinburgh Medical Journal, October, 1923.)

HENRI BOUTTIER

The unexpected death of Henri Bouttier has taken from France one of its most productive young neurologists. A young man, incessantly active, of unlimited energy and ambition, he always worked to the limit of physical endurance. While taking a few days of rest

in the mountains he developed a volvulus, and although operated upon apparently with success, he did not recover.

Bouttier early became interested in diseases of the nervous system. During the war, at the neurosurgical center of the 5th army, directed by Lecène, and later at the neuropsychiatric center at Épernay, he did excellent work on the pathology and neurophysiology of traumatisms of the brain and meninges.

Later, as *chef de clinique* in the service of Prof. Pierre Marie at the Hospice de la Salpêtrière, he became the devoted collaborator of his famous chief in a series of studies of great interest on the treatment of epilepsy, syndromes of disorientation in space, troubles of sensibility of cerebral origin, etc. In all of these studies he gave evidence of the most minutely accurate investigation, combined with critical judgment and the ability to transmit his ideas in a clear and concise exposition—qualities which have done so much to spread the renown of the neurology of France.

Great as was his interest in neurology, it did not hinder him from acquiring a well rounded knowledge of medicine in all its branches and such were his intelligence and energy that he was able to pass his concours as *médecin des hôpitaux* before the age of thirty-five.

He was just as indefatigable in the service of his friends as he was in the search of truth, and those who knew him well, as I came to know him, mourn not so much the brilliant physician as the earnest and devoted friend.

PERCIVAL BAILEY

NOTES AND NEWS

NEW NEUROPSYCHIATRIC SOCIETY

The physicians of the National Insane Asylum, at a recent meeting, organized the Mexican Society of Neuropathology and Psychiatry. Dr. Nicolás Martínez, director of the institution, was appointed President of the society and Dr. Norma, Secretary.

SUCCESSOR OF PROFESSOR PIERRE MARIE

Two new professors for the Faculté de médecine of Paris have been elected: Dr. Georges Guillaín in place of Pierre Marie, who has retired, and Dr. J. A. Sicard, in place of Rénon, deceased.

The twenty-eighth Congrès des médecins aliénistes et neurologistes of France and French-speaking countries is to be held at Brussels in August, 1924, with Professors Glorieux of Brussels and de Massary of Paris presiding. The secretary is Prof. A. Ley of Brussels. The questions appointed for discussion are "Mental Debility in Children"; "How to Study Disturbances in Speech," and "Adaptation of the Mentally Diseased to Their Environment."

At the third annual meeting of the Canadian Neuro-Psychiatric Association in Kingston, October 17, the following officers were elected for the ensuing year: President, Dr. Nelson H. Beemer, Mimico; Vice-President, Dr. James M. Forster, Whitby, and Secretary, Dr. F. S. Vrooman, Toronto.

N. B.—All business communications should be made to *Journal of Nervous and Mental Disease*, 64 West 56th St., New York.

All editorial communications should be made to Dr. Smith Ely Jelliffe, Managing Editor, 64 West 56th St., New York.

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ORIGINAL ARTICLES

NOTES ON THE NATURE AND ORIGIN OF THE CEREBROSPINAL FLUID *

By G. B. HASSIN, M.D.

ASSOCIATE PROFESSOR OF NEUROLOGY, COLLEGE OF MEDICINE,
UNIVERSITY OF ILLINOIS, CHICAGO

Of a great many neurologic problems of interest to clinicians, research workers and even general practitioners, the most obscure one is that pertaining to the cerebrospinal fluid. While there is hardly a practitioner that has not heard of the existence of this liquid, so extensively utilized for diagnostic and therapeutic purposes, there is hardly an investigator, however learned and brilliant, that knows exactly what the cerebrospinal fluid is, where it comes from and what is its function. Yet it dominates the attention of numerous workers in the fields of serology, physiology, pharmacology, embryology, and histology. It possesses an enormous literature. Not only short and extensive treatises, but even books have been devoted to this mysterious fluid. The majority of the contributions deal with its anatomy and physiology, those of Key and Retzius, Quincke, Weed and Becht being among the best. The studies of the foregoing workers brought forth some definite facts as to the seat, circulation and modes of escape of the cerebrospinal fluid. For instance, it is definitely established that it is lodged within certain cavities known as the ventricles of the brain and the cerebrospinal subarachnoid space. The former are situated within the hemi-

* A lantern slide demonstration before the Medical Society of the Mt. Sinai Hospital, Chicago, December 18, 1922.

spheres and the brain stem, the latter outside, forming an extensive space around the brain, cerebellum, pons and the spinal cord. It is commonly admitted that the inside cavities—the ventricles—communicate with the outside space by means of the foramina of Magendie and Luschka, situated in the fourth ventricle. Through these openings, the fluid flows from the fourth ventricle into the subarachnoid space where it circulates enclosed within the meningeal walls and their prolongations—trabeculæ. The walls consisting of the pia and arachnoid membranes, commonly called pia-arachnoid, are, like the trabeculæ, lined with mesothelial cells. In the neighborhood of the large sinuses, such as the superior longitudinal, for instance, the arachnoid portion is provided with prolongations invading the dura and known as the arachnoid villi or Pacchionian bodies (hypertrophied arachnoid villi of Weed).

The ventricles of the brain—the other important seat of the cerebrospinal fluid—are as noted situated within the brain substance. Their walls are lined with ventricular ependymal cells and the tuft cells of the choroid plexus which fills the ventricular cavities.

It is commonly assumed that the spinal fluid secreted and poured by the choroid plexus into the ventricles, flows from there to the third ventricle, aq. Sylvii, fourth ventricle, and then through the openings of Magendie and Luschka to the subarachnoid space. In the latter it is absorbed by the arachnoid villi. The fluid is also absorbed by way of the perineural spaces of some cranial nerves (olfactory, optic, trigeminal, hypoglossal). From the latter it reaches, through the tissue spaces, the lymph vessels of the neck and the general blood circulation. Such is the current teaching of the origin, seat, circulation, and the mode of absorption of the cerebrospinal fluid, a teaching based on physiologic, pharmacologic and similar experimental studies.

While some of the foregoing views, as for instance, the one pertaining to the seat, circulation and the mode of absorption of the cerebrospinal fluid are apparently firmly substantiated and universally accepted, the question as to the point of its origin is still a matter of dispute. Becht,¹ for instance, does not admit that the choroid plexus secretes the cerebrospinal fluid, the results of his experimental studies being in flat contradiction to those of equally competent and dependable workers.

Without going into detailed discussions of the methods employed in the studies of the various problems pertaining to the cerebrospinal fluid, I wish merely to present here a few facts obtained from studies

of histopathologic changes of the central nervous system. Instead of experimental methods, a histopathologic method is suggested together with a plausible explanation of the findings furnished by this method.

Histopathologic studies of the brain and spinal cord, including the subarachnoid space, ventricles and the choroid plexus, show that, in addition to the foregoing cavities—ventricles and the subarachnoid spaces—there are other spaces in the central nervous system which harbor the cerebrospinal fluid. Such cavities, countless in amount and microscopic in size, are situated within the walls of the blood vessels, in their adventitial layer. Known as perivascular, adventitial, or Virchow-Robin spaces, they are not seen under normal conditions, but in pathologic states, they are very prominent, being sometimes enormously dilated and packed with various pathologic elements (lymphocytes, plasma cells, fat-granule bodies, polyblasts, etc.). It is firmly established that the Virchow-Robin spaces are in communication with the subarachnoid cavity by which they are drained. In this manner the contents of the countless perivascular spaces of the brain and spinal cord are discharged into the subarachnoid space. There is some experimental evidence (Wislocki and Putnam)² that the ventricles are also in communication with these formations, that the latter thus are most likely drained, not only by the subarachnoid space, but also by the ventricles. If such be the case, then the flow of the cerebrospinal fluid must be away from the brain tissues and toward the foregoing cavities. Experimental as well as histopathologic facts seem to firmly establish such a possibility, that the direction of the flow is toward the subarachnoid space. But while the experimental workers claim that the point of origin of the flow is the lateral ventricles, or what is practically the same, the choroid plexus, the histopathologic facts do not wholly bear out such a view. It is hard to understand why the fluid originates only in the lateral, when the choroid plexus is also present in the third and fourth ventricles.

As to the direction of the current, the histopathologic facts are in full accord with those of experimenters. For instance, in a case of meningeal carcinoma³ the subarachnoid as well as the subdural space and the dura itself were intensely infiltrated with carcinoma cells. Yet, not a single cell was found in the brain substance itself. The same freedom of the brain from invaders was observed in a case of cerebrospinal meningitis.⁴ Here the pia was immensely infiltrated with plasma cells, and the subarachnoid space was densely packed with pus cells. Neither pus, nor plasma cells, however, penetrated

the parenchyma of the brain which was perfectly free from contamination by foreign elements.

These two instances demonstrate that were the flow of the cerebrospinal fluid away from the subarachnoid space and toward the brain, the latter would show the presence of pathologic elements. However, such *may* be the case under *certain* conditions. Weed,⁵ for instance, found granules of Prussian blue in the perivascular spaces of Virchow-Robin when he was injecting into the subarachnoid space a solution of iron ammonium citrate and potassium ferrocyanide under high pressure. In a case of meningeal carcinoma⁶ the subarachnoid space was so densely packed with carcinoma cells that they transgressed by mere pressure the pial barriers and landed in the superficial strata of the cerebrum. In tuberculous meningitis the subarachnoid space may be so intensely infiltrated that the superficial cerebral layers become involved as the result of the overflowing of the meninges and their spaces by pathologic elements.

In these instances of *intense* infiltrations of the subarachnoid space, the carcinoma as well as the tuberculous cell-bodies invaded the brain substance by way of the perivascular spaces of Virchow-Robin. This fact proves conclusively that the latter formations are in direct communication with the subarachnoid cavity.

An entirely different aspect is presented when the brain substance itself is involved. Experimental facts in this direction are furnished by Foerster.⁷ Injecting sterile india ink into the brain of the animals, he could locate it in the subarachnoid space.

Even more convincing are pathologic facts. For instance, cases of cerebral carcinoma⁶ in which the tumor masses were situated remote from an apparently normal pia, the subarachnoid space was distended and filled with a great variety of pathologic cell bodies. Among these were lymphocytes, polyblasts, fat-granule bodies (gitter cells), clusters of mesothelial cells, proliferated fibroblasts, and even cell bodies of distinctly epithelial character resembling carcinoma cells. The changes in the subarachnoid space were here evidently due to the discharge by the brain of its waste material into the subarachnoid space, where it provoked various proliferative and hyperplastic phenomena, outlined above.

The discharged waste, whether in the form of cellular elements or chemical substances, acts upon the pia-arachnoid and its linings (mesothelial cells) like any other foreign body artificially injected into the subarachnoid space. Essick,⁸ for instance, injected laked blood and found a remarkable reaction on the part of the mesothelial

cells which became enlarged, detached, clustered and transformed into definite and very active phagocytic cells (macrophages). Pathologic and experimental observations thus tend to demonstrate that the cell bodies enclosed within the subarachnoid space react in a certain manner against the presence of foreign or pathologic substances.

In some instances the reaction may attain extensive proportions. Thus a solitary tubercle of the spinal cord is always associated with a tuberculous meningitis.⁹ In some cases the tubercle is divided from the subarachnoid space by a strong and healthy pia. Yet the entire subarachnoid cavity and even the dura are found packed with products typical of tuberculous meningitis. The occurrence of the latter can only be understood by assuming that the tuberculous parenchyma of the spinal cord, drained by the subarchnoid space, carries with it infectious matter which sets up a lepto- and pachymeningitis. The reverse does not obtain, unless as noted, the pia-arachnoid is infiltrated too excessively.

Still more striking and convincing are the subarachnoid changes in degenerative conditions of the central nervous system (softening of the brain or spinal cord, multiple sclerosis, subacute combined cord degeneration and similar conditions). Numerous areas of degeneration packed with lipoids are scattered throughout the brain and cord, and separated from the pia-arachnoid by quite healthy portions of nerve tissue. Yet large amounts of the lipid substances are to be found in the subarachnoid space, enclosed within gitter cells, in its distended meshes or in the Virchow-Robin spaces. The latter are found to contain lipoids also throughout the brain substances, even in areas that are perfectly healthy and far away from the focus of degeneration. Originating in the latter, the lipoids travel by way of the perivascular spaces until they reach the place of their destination—the subarachnoid space.¹⁰

Quite unique are the findings in brain abscesses. Surrounded by a connective tissue capsule a brain abscess is sometimes divided from the subarachnoid space by a large tract of perfectly normal brain tissue. The capsule encloses large deposits of lipoids derived from the degenerated brain substance transformed into an abscess. The lipoids invading the capsule by way of the perivascular spaces, are carried by the latter to the subarachnoid cavity, where they can be located. As some infectious matter might be carried along with the lipoids, the resulting reactive phenomena will somewhat vary from those observed in ordinary degenerative conditions. They are represented by macrophages packed with broken up polymorphonuclear

cells, pigment granules, degenerated lymphocytes, as well as numerous gitter cells filled with lipoids—all these elements crowding the distended meshes of the pia-arachnoid and the perivascular spaces of the cerebral and meningeal blood vessels.

Equally extensive and intensive reactive phenomena can be found in cases of so-called cerebral porosis¹¹ and calcification of cerebral vessels.¹² In the former the white cerebral substance is filled with cavities produced by an invasion of aerogenous bacilli; in the latter (calcification) it contains large deposits of lime salts.

In the subarachnoid space of the cerebral porosis brain were found erythrocytes, lymphocytes, plasma cells, polyblasts, gitter cells, amorphous granules of blood pigment, clusters of proliferated mesothelial cells and gas bacilli.

In the case of calcification of the cerebral vessels the tissue spaces were infiltrated with lime salts, whole areas including the parenchyma, the blood vessels and capillaries being transformed into lime substances. Absorbed by the Virchow-Robin channels and landed in the subarachnoid space, they provoked here violent reactive phenomena in the form of pronounced hyperplasia of the pia-arachnoid.

The frequent occurrence of hemorrhagic foci in the brain affords unusual opportunities for studying the relationship between the subarachnoid and the Virchow-Robin spaces. Both are found filled with blood pigment which can be demonstrated not only in postmortem specimens, but also *intra vitam*, in the spinal fluid. While in embolism or thrombosis of the cerebral vessels, the spinal fluid always gives a negative benzidin reaction (provided the spinal puncture is devoid of traumatic blood), in cerebral hemorrhages, however old, this test is always positive.¹³

The few quoted instances taken from the studies of histopathologic changes of the central nervous system are quite convincing in that they prove that the subarachnoid space is a receptacle for the waste products discharged by the parenchyma of the brain and the spinal cord, that the contents of this formation are wholly derived from the central nervous system. The contents—under pathologic conditions—whether blood, lime salts, lipoids, tuberculous matter or carcinoma cells, etc., can be demonstrated as such under the microscope. In some instances they can not be seen, but can be demonstrated indirectly. For instance, in lead encephalitis,¹⁴ proliferative phenomena obtain in the mesodermal tissues (blood vessels, capillaries) because of the chemical action of lead salts. Assuming the latter to be present in the tissue fluids of the brain, they are carried through the

perivascular spaces to the subarachnoid cavity and cause there reactive phenomena, such as hyperplasia of the pia, proliferation of the mesothelial cells with new formation of capillaries, etc.

Pathologic changes, however, are to be found not only in the subarachnoid and the spaces of Virchow-Robin, but also in the third container of the cerebrospinal fluid—the ventricles and their enclosures, the choroid plexus. The tuft cells of the latter show interesting changes depending upon the character of the morbid condition. In so-called multiple softening of the brain,¹⁵ in the degenerative condition known as *polioencephalitis superior and inferior*,¹⁶ in multiple sclerosis,¹⁶ *myelomalacia*,¹⁷ the tuft cells are invariably filled with fat-like, lipoid, substances. Normally present in almost every choroid plexus, under foregoing pathologic conditions, they are found in excessive amounts; in some instances, the tuft cells are actually transformed into typical fat-granule bodies (*gitter cells*). The latter thus may be found not only in the Virchow-Robin spaces of the cerebral vessels, not only in the subarachnoid space, but also in the ventricles where they are taken or picked up by the tuft cells of the choroid plexus.

Still more convincing was a case of a hemorrhagic focus located in the tegmental region of the pons,¹³ where it was separated from the fourth ventricle by an area of normal brain tissue. Despite this, deposits of hemoglobin were found not only in the subarachnoid space, but also in the tuft cells of the choroid plexus of the fourth ventricle. In cases of cerebral carcinoma,⁶ the choroid plexus showed marked hyperplasia of the connective tissue and other changes which can be explained only on the basis of its coming in contact with a fluid derived from a contaminated brain.

In studying choroid plexus changes, one should bear in mind the plexuses of the lateral, third and fourth ventricles, and the necessity of studying them separately in a great number of sections. For some sections may show no manifest changes whatever, either by mere accident, or for reasons I cannot discuss here.

Were the choroid plexus an organ of secretion of the spinal fluid, it would not be possible to understand its behavior under the pathologic conditions mentioned. Nor would it be possible to understand the pathologic phenomena obtaining in the subarachnoid and the Virchow-Robin spaces. Assuming, on the other hand, the point of origin of the cerebrospinal fluid to be in the cerebral or spinal cord tissue spaces, the pathologic phenomena outlined above can be understood and logically explained. Originating in the tissue spaces, the

fluid comes into close and immediate contact with the tissue elements, it takes up and absorbs the products of their metabolism, or abnormal deposits (lipoids, tubercles, blood, lime salts, etc.), discharging them into the subarachnoid space and the ventricles. The spinal fluid thus viewed must in each case be chemically, serologically, and histologically different, depending upon the chemical, histologic and other conditions of the parenchyma of the central nervous system. The presence or absence of Lange test, the positive or negative Wassermann reaction, the presence or absence of the globulin and similar reactions, can be understood only on assumption that the cerebrospinal fluid is a direct product, a part, as it were, of the nerve tissues themselves. The correctness or plausibility of such a view is certainly borne out by histopathologic facts which cannot be understood nor explained by the secretory hypothesis of this substance. Originating within the central nervous system and discharged by way of the Virchow-Robin spaces into the subarachnoid chamber and the ventricles, it is absorbed, in the former by the arachnoid villi, in the latter by the choroid plexus. Both the arachnoid villi and the choroid plexus appearing automatically, at least under some pathologic conditions, alike, are organs of absorption of the cerebrospinal fluid.

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THE SKIN AS A FETICH; CASE REPORT

BY LEON EMILE DUVAL, M.D.

SENIOR ASSISTANT PHYSICIAN, WORCESTER STATE HOSPITAL,
WORCESTER, MASS.

From time to time the fetichist appears in the literature of psychiatry and legal medicine. A few writers like Forel and Kraft-Ebing have collected cases, but they are sufficiently uncommon to warrant further case reports. They are sometimes not recognized as having a relation to the psychosexual life, especially the types which collect handkerchiefs or shoes, or whose fetiches are not usually thought of as having a sexual significance.

Like the psychoses and other perversions of the psyche, fetichism may be thought of as an exaggeration or distortion of usual mechanisms. We know that for the average individual, the sex act must be preceded by certain stimuli, psychic and physical. Erotic desire is physiologically aroused by the sight of the sex object, or by the complex of ideas associated with the object. In many normal subjects the sex expression can only be aroused by a certain type of sex object, or under certain surrounding conditions. This mechanism is essentially that of a conditioned reflex. Carried to extremes, we have a certain type of fetichist, for example the man who is potent only with a deformed woman.

The handkerchief fetichist, the shoe fetichist and their ilk are still further developments of this mechanism. The usual starting point for such a case is an initial sex experience in which the object which is later to become the fetich stands out prominently in the experience so that it acquires a strong affective value. The sex stimulus, which has not previously been fixed, becomes associated not with the sex object as a whole, but with some particular attribute of that object. Therefore sex pleasure will be initiated not by the usual stimuli, but by the object which acquired affective value in the initial experience. This seems to be the explanation given by most writers. It is assumed that the reader is familiar with the hypotheses of Freud as to the development of the psychosexual life. He explains the neuroses as resulting from faulty development along this line, and many other psychic syndromes have been explained by the same hypotheses. It seems possible to the writer that some cases of

fetichism may be explained by a failure to reach normal heterosexual levels, or are abortive developments of the usual mechanism.

The following case is presented as an example of this, and also because I have failed to find in the literature a case in which visual and tactile stimuli from the skin of a love object of indifferent sex constituted in themselves a fetich capable of producing full sexual satisfaction. If I have wrongly interpreted this case, I stand ready for constructive criticism.

A. B., male, of American birth, aged thirty-five, admitted to Worcester State Hospital in March, 1922. The family history showed nothing of importance. There were no unusual events during pregnancy, labor or early development. From childhood his vision had been somewhat defective. On this account he had some difficulty with his studies, and because of his rather peculiar physical appearance he was the butt of his schoolmates' jokes. He was a younger son of a New England farmer, and an older brother was given control of farm affairs. Patient remained on the farm from a sense of duty to his parents, although his ambitions lay in other fields. He occupied the position of servant more than that of a member of the family, as he was never consulted in the affairs of the farm, his brother received the profits, and he was given only his clothing and tobacco for his labor. For years he has felt that he was not wanted in the family, and built himself a little cabin on the edge of the farm. This cabin brought him nearer his neighbors and partly satisfied his desire for the company of others. People were kind to him, but did not receive him as an equal.

He received no sex instruction, and for several years was addicted to masturbation. At twenty-four he began to consort with prostitutes at times. However, his sex life was not satisfactory, and it was not until the age of thirty that his sex life became at all crystallized.

About this age he noticed an attraction towards boys of ten or twelve manifested by a desire to massage their chests and backs, and to have them perform the same act upon him. During the process he would experience strong erotic feelings. The sight of the boys' bare skin was pleasurable, but the sensation of touch was more so. For several years he managed to have the neighborhood boys visit his cabin for this purpose. He also had an adult male friend who would allow him to do the same things. However, this man made overtures suggesting fellatio, which greatly disgusted the patient. Patient's cabin was near a cheap pleasure park, and prostitutes used to come to his cabin. He found that before he could perform coitus, he had to look at and rub the woman, otherwise he was impotent. Coitus itself was not particularly pleasurable to him. At times he would be impotent both with men and women, due to excessive fatigue, or masturbation into which he occasionally lapsed when he could find no sex object.

It is worthy of note that patient has never made sex approaches

to others, fearing that he would make overtures to some one who would refuse him, and perhaps expose his peculiarities. For years he has lived in fear of discovery by his parents and the neighbors. A conflict with the mother of one of the boys threatened to bring out these things and patient went into a confused state in which he stabbed himself. A few days residence in this hospital were sufficient for him to return to his usual mental state, and he has been back in the community for a year. Physically he was poorly developed, had reddish hair and albino eyes, infantile genitalia, asymmetrical skull, and general blunting of tactile sense. Laboratory findings were all negative. The patient had an unusually good insight into his own peculiarities.

The most important feature of this case is that the individual, after several years of masturbation and unsatisfactory heterosexual experiences discovered that full sex satisfaction could be obtained from having his skin massaged, or by rubbing the skin of another, either man or woman. This form of pleasure is exceedingly infantile. The first pleasure-sensations which the infant learns are those of satisfying hunger and emptying the emunctories. A little later he obtains pleasure from the bath and the rubbing and massaging which go with it. In later periods of development the foci of pleasurable stimuli become localized in the pelvic region, and, in women, in the nipple region. Lesser foci are found in other parts of the body. The localization in the genital region becomes marked at puberty.

Our patient apparently remained in that infantile stage where pleasure stimuli were referred to the skin in general, as even in adult life he never obtained real sex pleasure from the usual stimuli. He failed to develop that differentiation of sensory areas by means of which the foci for sex stimulation become localized in the generative organs. This deep-seated developmental defect can hardly be modified, being part of a general constitutional infirmity. Under conditions of stress he will undoubtedly develop other psychotic episodes. However, in a little country community, protected by relatives, he may continue to be for an indefinite period an economic asset, and is not particularly dangerous to community morals.

A STUDY IN DIFFERENTIAL DIAGNOSIS

By W. W. YOUNG, A.B., M.D.

RESIDENT PHYSICIAN, HIGHLAND HOSPITAL, ASHEVILLE, N. C.

In the past certain disease entities have been characterized by and diagnosed upon the basis of so-called syndromes. But as the studies of postmortem material became more elaborate, more numerous, and more exhaustive; when we included a consideration of the period of incipency, and as our methods of clinical examination and study became more exact and exacting, the reliance upon the existence in part or as a whole of these syndromes approached more nearly the vanishing point. When we could hypothecate the existence of such and such a syndrome and diagnose only on the basis of its existence how simple a question differentiation was. But when study reveals the possibility of the existence of a specific disease without the well defined presence of any of the original members of the syndrome, then does differentiation become a matter of more and more complex study.

The syndrome of multiple sclerosis was at one time thought to be well-nigh infallible. The diagnosis was made when there existed at least two of the classical triad: nystagmus, scanning speech, and intention tremor. Later when the use of the ophthalmoscope and the examination of the eye in general became more frequent and more refined, there was added to this triad the condition of the optic disc, showing temporal pallor or other signs of a degenerative character. Then later the loss of the abdominal reflexes was found to be a very prominent symptom, so that this finding began to bulk large in the estimate of many in making a differential diagnosis. So closer and closer study, beginning with the more incipient stages up to the lethal period, has changed our conception until the original Charcot triad has faded into an insignificant position. It may not even exist in some cases which are still very definite. And now the investigations of the Association for Research in Nervous and Mental Diseases have brought out the fact that it is only by study of the mode of onset and the course of the disease, coupled with a careful consideration of the clinical findings that one may make a worth-while differentiation. Even then it is a matter of extreme difficulty in many instances,

The case which we shall use as the basis for this discussion has offered many difficulties. Even at the present time there is a difference of opinion as to the actual diagnosis. Two distinct diagnoses have been made by men of ability and discernment and so we feel that a study of the arguments for and against each position will be more or less stimulating. The one weak point in the discussion is that we are not able to offer postmortem confirmation of either view since the patient is still living. However, this fact does not detract from the interest of the case itself.

Mrs. P., age fifty-three years, first came to this hospital in 1916 with a well-defined psychoneurosis in which she showed well marked emotional instability. Her family history revealed a father who died at the age of sixty-seven of cirrhosis of the liver and organic heart disease; and a mother who died at the age of sixty-three of "paralysis." There was a brother who died of apoplexy while a living brother and a sister, each has had a "slight stroke of paralysis." The patient's own history shows her to have been far from robust as a girl. In 1898 she had a miscarriage following a fall from a hammock and this in turn was succeeded by several others, the duration of each pregnancy not being known. In 1913 there was an operation at which a fibroid uterus and probably her ovaries were removed. The illness under discussion began with the complaint of too readily fatiguing. The examination at that time revealed no marked organic abnormalities. The tendon reflexes were all exaggerated; the abdominal reflexes were present; and the pupils were somewhat sluggish in reacting to light. The urine intermittently showed a slight trace of albumin and on one occasion rare hyaline casts. After a stay of some months the patient's symptoms improved sufficiently for her to return home in comfort.

She was readmitted June 28, 1921. For a number of months she had found herself losing strength in addition to the former sense of readily fatiguing. There was a vague sense of awkwardness and some suggestive difficulty in pronunciation. All this in a short time was followed by the appearance of a typical hemiplegia with the classic signs of organic lesion. Examination showed the pupils to be somewhat contracted and the light reflexes of small excursion. All the tendon reflexes were increased and there was defective coördination of the extremities though there was no outspoken intention tremor. The urine showed the same condition as before and the blood showed hemoglobin 75 per cent, and RBC-3,550,000 per c.mm.

In February, 1923, a note was made as follows: "Pupils are equal and even, somewhat contracted though not marked, and react to light and accommodation, consensual and direct, though the reaction to light shows a limited excursion. Extraocular movements are normal. The other cranial nerves are negative except that there is some speech disturbance, a very slight slurring, although she reproduces the usual test syllables with fair accuracy. There is a spastic

paresis of the right arm and leg and all the deep reflexes are exaggerated, more particularly on the affected side. The abdominal reflexes are absent on the parietic side and hyperactive on the other. Babinski, Oppenheim, and Gordon not clean-cut but positive." Ophthalmoscopic examination showed the eye grounds to be normal with no temporal pallor or any signs of degeneration. The visual fields were not taken. The blood Wassermann was negative on repeated examinations, as was that of the spinal fluid. The spinal fluid shows 9 cells per c.mm.; globulin, Ross-Jones, double plus; Pandy, double plus. The colloidal gold and colloidal mastic were negative on two examinations. A third colloidal gold showed a suggestive luetic curve: 1122100000. Since this time the patient's condition has remained stationary except for a more recent note which shows a more pronounced speech defect.

Looking at the case from this isolated viewpoint in cross-section without a knowledge of the previous examinations, history, and conclusions, one probably would unhesitatingly feel that one was dealing with a simple hemiplegia due to a lesion within the internal capsule of the brain without any serious thought of the necessity for differentiating from any other type of disease. As it was, the prejudice was all in favor of multiple sclerosis because of the previous opinion and the expectations were directed to this end. Taken from this viewpoint it is necessary to coördinate the findings and argue to a logical conclusion.

In the first place we have the history at the time of her first admission in 1916 of easy fatiguability which gradually grew in proportions with periodic remissions until the development of a hemiplegia. And this is a most salient point in the history of cases of disseminated sclerosis. There is always a history of easy fatiguability with transient weakness, this coming and going at first but gradually though progressively growing worse. There are distinct remissions which mislead the patient and the physician as well into thinking that they have been rid of the symptom. Then there is the development just prior to her second admission of a vague sense of awkwardness and slight subjective difficulties with her speech. At this time examination revealed some pupillary abnormality with increased tendon reflexes and a distinct though not great distal incoördination.

Examination two years later showed the loss of abdominal reflexes on one side. There was a slight remission of the incoördination and the speech difficulty, the latter showing a worsening some two months later, however. Babinski was positive though not a clean-cut reaction. In addition to these physical findings the blood

Wassermann was persistently negative even after giving, in February, 1923, a provocative salvarsan injection. The spinal fluid Wassermann was also persistently negative, whereas the cell count and globulin showed the presence of an inflammatory or degenerative process. The colloidal gold curve on only one occasion was positive and this time not definitely so but approached a luetic curve. This type of curve is often found in cases of multiple sclerosis. Psychic symptoms, emotional instability, predominated at the outset. This type of emotional reaction quite frequently is attendant upon a disseminated sclerotic process.

These findings together with the remissions in various symptoms which were not marked, however, argue quite strongly for a diagnosis of multiple sclerosis. When we consider the history of onset and the general course of the disease our belief is strengthened. And taking the disease from the standpoint of its pathology: the presence throughout the central nervous system of scattered plaques of sclerosis, following no definite law of distribution, we find our picture can be fitted in quite readily.

On the other hand there is another viewpoint which looks equally as plausible. First, there is the family history which points to a familial relative susceptibility on the part of the vascular system and more particularly the neural vascular system. We find the mother and one brother succumbing to the effects of degenerative changes in the blood vessels of the brain and also a brother and a sister suffering from the effects of such a condition which did not prove lethal. Again, it is suggestive, though nothing more, that the patient herself has been the victim of several miscarriages which would argue, if there were additional testimony, for the presence of a syphilitic infection. Now if we discard everything but the findings in the patient herself, we find nothing which cannot readily be explained on the theory that she had a vascular lesion in the brain, most likely in the region of the internal capsule. The onset is not incompatible with this supposition. There is a most definite hemiplegia with a spastic paresis of the arm and leg. There are increased tendon reflexes on the side of the lesion. There is a loss of the abdominal reflexes on this side. The Babinski is positive. And the slight speech disturbance with its tendency to remissions and the emotional changes are not incompatible with this idea. It is true that the Wassermann in both blood and spinal fluid is persistently negative. But we have the other evidences that there is an inflammatory process going on in the brain and on one occasion there was a suggestion of a luetic curve

in the colloidal gold test. On the other hand many cases exhibiting frank syphilitic neuropathology persistently gave a negative Wassermann.

We must confess that the arrival at an unquestionable differential diagnosis seems to us well-nigh impossible. When we consider the history with the possibility of onset back in 1916 with gradual development beginning with emotional instability and easy fatigability, this growing to actual weakness although with remissions and progressing to an hemiplegia with dysarthria, incoördination, and loss of abdominal reflexes on one side, we seem to have most convincing arguments for multiple sclerosis.

Whereas there is one weakness here: in the report of the Commission Dr. Sachs says, "May I say one thing about this reflex (abdominal reflex)? The value of it is extremely great in this way: that if we find it associated in other conditions with other reflexes, for instance, in case of hemiplegia, the absence of it on the hemiplegic side and the marked presence of it on the other is very characteristic. And the one thing that is said about the diminution of the abdominal reflex in multiple sclerosis is that it is almost uniformly symmetrically affected." And here, too, we are left at loose ends by the use of the term "almost." However, arguing from this dictum as a starting point and considering all the factors in this case the more plausible conclusion seems to be that we are dealing with a vascular lesion in the internal capsule most probably of syphilitic origin. And into this hypothesis there is no finding either in the history of the case, the physical examination, or the biologic reactions which cannot be fitted. Whereas the most potent argument against it is purely negative in the absence of a positive Wassermann in blood or spinal fluid.

We have found this case most stimulating and have used this as an excuse for presenting it here in the hope that others may find it equally so. At any rate it illustrates very nicely the difficulties of final and conclusive differentiation in the absence of clear-cut syndromes. It would appear at first glance that the growth in our knowledge and in the refinement of our methods acts as a hindrance to forming accurate conclusions instead of being a help. But on the other hand we know that through the means of exhaustive study we do arrive at more accurate diagnoses than ever before and that in the past many were incorrectly diagnosed as shown by Cabot's statistics at the Massachusetts General Hospital after comparison of the post-mortem with the clinical findings.

The lessons which we have learned from the study of this case are valuable to us. It teaches us, first, that in any branch of internal medicine and more particularly in neuropsychiatry it is disastrous to approach the case from the antiquated "cross-section" viewpoint but we must study it in "long-section" over an adequate period of time and that, too, until we feel we have exhausted our resources. Secondly, differentiation is not the simple thing which it appears often on superficial approach, and every resource known to science should be used in every case without preconception and with an unbiased attitude. For so often one single point will be the fulcrum on which the lever rests which is to pry out the correct interpretation. And, lastly, in the case of disseminated sclerosis itself, we must assume the attitude prompted by an understanding of the essential pathology, and not think we are going to get a clear-cut picture of syndromes but a very heterogeneous one, resultant upon the distribution of the sclerosis in the particular case under study. Therefore we must study every case exhaustively in "long-section," using all the methods at our disposal and interpret our findings on the basis of reasonable interpretation rather than upon preconceived ideas of the relation of the symptomatology.

EPIDEMIC (LETHARGIC) ENCEPHALITIS *

A CLINICAL STUDY OF THIRTY-FIVE CASES GATHERED DURING THE RECENT EPIDEMIC, INCLUDING SOME RETROSPECTIVE CASES

BY MAX H. WEINBERG, M.D.

ASSISTANT NEUROLOGIST, WESTERN PENNSYLVANIA HOSPITAL,
PITTSBURGH, PA.

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A nurse, case 17, showed more spectacular psychoneurotic symptoms than the preceding case, but recovered fully in response to suggestion. From the very onset she had some psychoneurotic manifestations, particularly a feeling of bigness, but the major attack developed on the sixth week, after the organic symptoms subsided. For close to two months she insisted that she was blind and unable to walk. The room had to be kept in utter darkness, and the patient employed all means to shut out any stray ray of light. The blinds were drawn, and pillows placed with weights to keep them down tight. She, herself, wore dark glasses and had towels wrapped around them. But, under powerful suggestion she cleared up in two days and walked around by herself. In about a month she cleared up completely. A note from her twenty months later stated that she had no difficulty with her eyes or stomach, and her only complaint was that she did not have the endurance she had before she took sick.

The other two, both retrospective cases, 29 and 31, complained of irritability, extreme restlessness, and one, case 31, of having the "blues" frequently.

Only one of the four, case 17, ever had psychoneurotic symptoms before this sickness.

Convulsions. But few observers mentioned this symptom and then only in connection with children affected. Alexander and Allen, and Barker, do not mention convulsions. Comby (21) mentions that convulsions, spasms, and unconsciousness for twelve to twenty-four hours are common. Findlay and Shiskin (22) merely mention convulsions without specifying frequency. In this series there were four cases, 11 per cent, that had convulsions during the acute stage,

* From the service of Dr. W. K. Walker.

although in two of them, 11 and 12, this could not be definitely established, but we were certain of a period of unconsciousness at the very onset of the disease. One, case 20, fell unconscious while at work. The mill physician diagnosed fracture of the skull. A clear history could not be obtained. One, case 11, fell unconscious while at play. In two cases, 2 and 7, the convulsions occurred during the disease, and they were of a typical grand mal attack. In case 2, the first convulsion came on in the eleventh day of the disease, and he had three in rapid succession. The next day he was brought to the hospital. He again had a convulsion during the third night, and one on the fourth, biting his lower lip severely during the last one. This patient never had convulsions before, but his family physician related that eight weeks before the onset of the disease he had a short period of confusion following his mother's death. It would seem that at least there was some instability in this individual.

In case 7 the convulsions came on with a hemiplegic attack after the patient was sick for about six weeks with what was believed to be influenza, and lethargy of four weeks duration. He had but two convulsions.

Only one of these four, case 11, was a child.

Hyperidrosis. This symptom was observed in three cases, 9 per cent. It manifested itself differently in each one. In case 2, the sweating was limited to the lower limbs. It came on during the fifth week of the disease and lasted but forty-eight hours. It was very discomforting to the patient.

In case 9, it came on about thirty-six hours ante-mortem and was general. This was probably more dependent on the general toxic condition than on any special localization. Of extreme interest was the unilateral hyperidrosis in case 6. This developed several days after the appearance of the myoclonic movement synchronous with respiration which was increased.

In discussing the myoclonic movement, it was pointed out that the lesion was probably in the medulla. This symptom further tends to prove this localization. Depisch (23), in discussing the pathology of the vegetative nervous system, arrives at the following conclusion; "The decussation of the path for the vasomotors and the sweat glands of the trunk and extremities appears to be located in the upper segments of the medulla oblongata." The coexistence of these two symptoms in this case would tend to prove their location in the medulla where structures are packed closely together and a small lesion would be apt to produce varied symptoms.

The sweating was extremely discomforting to the patient. It lasted for the two months that he remained in the hospital. I then lost track of the patient and am unable to say when or whether it cleared up.

Cephaloptosis. This symptom was reported in children only, but in this series it was present in two children and one adult, three cases, 9 per cent, in all. In case 6, the adult, the cephaloptosis developed late in the disease during the fifth month. In case 21, a child of five, the symptom came on immediately after he left the hospital, and when seen three months later it was very marked. At the reëxamination, eighteen months later, the symptom had disappeared.

In case 33, a retrospective case, examined eighteen months after the onset of the illness, I could not determine when the symptom appeared for the first time, but it was marked at the time of the examination.

Endocrine Glands Involvement. Howe (24) reported four cases of the disease that showed clinical manifestation of endocrine involvement. Three cases, 9 per cent, in this series, showed definite endocrine conditions; one, case 11, pituitary disturbance, and two, cases 18 and 24, thyroid disturbance. In case 11, the pituitary symptoms manifested themselves during the latter part of the third month. Up till then, the patient, a child, was extremely emaciated and took food with difficulty, but in about a week he gained over twenty pounds and enough food could not be supplied. He soon commenced to walk and he would go around among the other children and eat all the left-overs, and even grab their food.

In case 18, acute thyroiditis developed during the fifth week of the disease. The patient complained of pain along the left sternocleido-mastoid muscle. The thyroid, previously somewhat large, became enlarged greatly, and the pulse rose to 110. This condition lasted for a week only. In case 24, there was a sudden enlargement of the thyroid three days before his death. The patient was so desperately ill, however, that the condition was not studied sufficiently. An autopsy could not be obtained.

Greasy Face. A peculiar greasiness of the face was present in three cases, 9 per cent. This usually disappeared late in the disease, although traces of it limited to one part of the face may persist for a long time. In case 4, the nose was greasy over two years after the illness.

Dysphagia. Only twice was this symptom observed, in cases 8 and 9, 6 per cent. In case 8, it developed during the third week of the

disease at the same time that the labial paralysis came on. It lasted but a week. In case 9, the dysphagia appeared three days before the patient's death.

Choreic Manifestations. Two cases, 6 per cent, showed choreic manifestations at the onset. In case 8, the diagnosis of chorea was made by the attending physician before the patient was sent to the hospital. The movements were typical of Sydenham's chorea. They cleared up in about five days. In case 26, the choreiform movements were of a violent nature, almost throwing the patient out of bed and doubling her up so that only the buttocks touched the bed. It was more like an electric shock, and each one was accompanied by a shrill cry. These movements ceased at the end of forty-eight hours.

Spasmodic Tic. In two cases, 6 per cent, spasmodic movements developed. In one, case 8, it manifested itself first during the tenth week of the disease when the patient was apparently recovered. It began as a movement involving the lower left facial and sternocleidomastoid muscles, occurring about once a minute. This, like the myoclonic movements, was preceded by pain for a few days. The patient left the hospital in two days and was observed in the outpatient department for the next six months. The movement spread to the left arm and leg during the next month or so and changed its character. Every movement pulled the head downwards, raised the shoulder, and pulled the leg upward. This was more of the type of a torsion spasm of the dystonia musculorum type.

In case 27, a retrospective case, that was not seen until the fourth month of the illness, the spasm of the right neck muscles and lower face came on during the second week and retained its characteristic tic movement. It is noteworthy that this patient had respiratory difficulty and he may have been somewhat analogous to case 6 who had myoclonic movements of the fingers of the right hand synchronous with respiration.

Sensory Changes. Although carefully searched for, sensory changes were observed in but two cases, 6 per cent. In case 24, there was hyperesthesia of the inner border of the legs and trunk for a few days during the fourth week of the disease. In case 31, a retrospective case, hyperesthesia of the left face was observed fifteen months after the onset of the illness.

Contractures. In one case, 11, 3 per cent, there was contracture in flexion of both legs for about three weeks during the height of the illness. This symptom disappeared completely.

Conjugate Deviation. Paralysis of conjugate dextroversion was

the first symptom noted in one case, 11, 3 per cent, immediately after the attack of unconsciousness which ushered in the disease. This symptom cleared up after about three weeks.

Arthritic Involvement. In one case, 23, 3 per cent, there developed inflammation of the right great toe. There were also milder inflammatory changes in other small joints. The inflammation subsided in a few days.

Temperature, Pulse, and Respiration. All of the acute cases showed increased temperature during their illness, especially early. The temperature is a fairly safe guide as to the severity of the condition. This is especially true of cases with extremely high temperature. Only one patient who had a temperature of 105 recovered. In all the three cases that died there was a marked ante-mortem rise, 105 in case 9, 106.5 in case 14, and 107.2 in case 24.

The pulse usually keeps up with the temperature. In only three cases did the pulse keep high in spite of the normal temperature, but there is a ready explanation for these exceptions. In one, case 18, there was a sudden enlargement of the thyroid which increased the pulse rate from 90 to 110. In two cases, 15 and 19, the development of marked psychoneurotic symptoms brought about the increased pulse rate independently of a high temperature. The respiration usually kept pace with the temperature, but was more apt to be normal. In the three cases that terminated fatally, the respirations were very high at the time of death.

Spinal Fluid. Of the twenty-six acute cases, three (cases 2, 18 and 26) had a cell count below five. All the others showed an increase from 6 up to 162 per c.mm. The cells were 90 to 95 per cent small lymphocytes. Plasma cells were occasionally encountered.

The globulin reaction was negative in five, weakly positive in seven, and positive in fourteen cases.

The colloidal gold test was done in twenty-two cases and only six showed any change, three of which were fairly marked.

The Wassermann test was negative in all but one, case 12, which was reported as slightly positive.

No fluid was normal in every respect. Even the five that had no globulin reaction showed other pathological features. All were under great pressure, two had an increased cell count, and one had a high sugar reducing power. Unfortunately, this latter test was done in cases 26 and 32 only. In connection with this, it is interesting to note that in retrospective case 32, the sugar reducing power remained high after two years and that it is the only pathological feature

remaining. During the last few months, we had several post-encephalitic patients, not reported in this series, and the fluid of every one of them shows an increased sugar reduction quality.

The urine quite frequently shows some alteration, but usually is not of sufficient importance. Occasionally changes are so marked that the uninitiated may make a diagnosis of uremia, as happened in case 20, but this occurs very rarely.

TABLE 1
CEREBROSPINAL FLUID FINDINGS

Case No.	Cells per c.mm.	Glob.	Wass.	Col. Gold.	App.	Remarks
1	8.5	Pos.	Neg.	0000000000	Clear	
2	1.	Ft. trace	"	0000000000	"	
3	24.	Sl. pos.	"	0000000000	"	
4	12.3	Sl. pos.	"	0000000000	"	
5	45.	Weak pos.	"	0012000000	"	
6	11.5	Weak pos.	"	1123210000	"	
7	22.	Pos.	"	0011000000	"	
8	21.	Weak pos.	"	0000000000	Cloudy	
9	14.	Neg.	"	0000000000	Clear	
10	12.	Trace	"	0000000000	"	
11	81.	Positive	"	0000000000	"	
12	6.	Pos.	Sl. pos.	0000000000	"	
13	162.	Heavy pos.	Neg.	0001100000	"	
14	18.	Pos.	"	1234510000	Turbid	
15	87.	Pos.	"	0000000000	Clear	
16	22.	Weak pos.	"	0000000000	"	
17	41.	Pos.	"	0000000000	"	
18	2.	Neg.	"	0000000000	"	Fluid under grt. pres're
19	10.	Neg.	"	0000000000	"	" " " "
20	12.	Sl. pos.	"	0000000000	Xanthocr.	
21	21.	Pos.	Not done	Not done	Clear	
22	4.	Neg.	Neg.	" "	"	
23	10.	Pos.	Not done	" "	Turbid	
24	50.	Strong pos.	Neg.	" "	Clear	
25	65.	Pos.	"	" "	"	Greatly increased pressure
26	2.5	Neg.	"	0000000000	"	Sugar 79 mg. per 100 c.c.
32	1.	Neg.	"	0000000000	"	Sugar 74 mg. per 100 c.c.

The mortality was unusually low in this series. Only three patients died, 11.5 per cent, computing it on the basis of the acute cases only.

Diagnosis. With increasing knowledge of the condition, this should be comparatively easy. I would emphasize the importance of being on the lookout for these cases, especially during the winter season, and particularly when the so-called "Flu" cases are common.

Any acute infectious condition that shows puzzling symptoms especially of a neurological nature that do not fit in very well with the ordinarily well-known diseases should at least suggest encephalitis, and a spinal puncture should be done.

TABLE 2
VARIOUS DIAGNOSES MADE IN THESE THIRTY-FIVE CASES BEFORE TRUE
CONDITION WAS RECOGNIZED

Case No.	Original Diagnosis	Case No.	Original Diagnosis
1	Undetermined	19	Encephalitis
2	Epilepsy	20	Fracture of Skull
3	Brain Tumor	21	Undetermined
4	Nephrolithiasis	22	Hysteria
5	Undetermined	23	Undetermined
6	Typhoid	24	Paramyoclonus
7	Influenza, Hemiplegia	25	Meningitis
8	Chorea	26	Encephalitis
9	Influenza	27	Undetermined
10	Undetermined	28	Undetermined
11	Fracture of Skull	29	Meningitis
12	Alcoholism	30	Encephalitis
13	Undetermined	31	"Flu" pneumonia
14	Undetermined	32	Undetermined
15	Encephalitis	33	Undetermined
16	Acute abdominal cond.	34	Psychoneurosis
17	Bell's palsy	35	Encephalitis
18	Encephalitis		

Table 2 was prepared for the purpose of showing the many conditions that encephalitis was mistaken for. At the present day, I have a case of encephalitis that was diagnosed as influenza. When the patient developed delirium after a sudden rise and equally sudden subsidence of temperature, the attending physician diagnosed hysteria. A careful neurological examination revealed many symptoms suggestive of encephalitis, and the subsequent course of the disease proved it.

RESIDUA AND SEQUELÆ

I have grouped the cases that I reëxamined and those that have not come to my attention till long after the initial illness, under two terms, namely, cases showing residual symptoms, and those showing sequelæ. In the first group I included all those cases that could either return to their previous occupation or adjust themselves to a new situation, but still showed symptoms. In the second group, I included all those cases that remained badly crippled physically, or mentally, or both.

I have included all the cases, both acute and retrospective, in the same group for the study of end-results, although on the surface it may seem that this renders the prognosis more serious. The twenty-six acute cases that I am reporting represent but a small part of all the cases seen by me during that period, but unfortunately, owing to the fact that most of them were being treated in private homes, observations were not recorded. A sufficient percentage of cases of that group developed serious sequelæ, and this to my mind more than offsets the addition of these six retrospective cases.

In all, seventeen cases are considered. Seven of these are residual, and ten show marked sequelæ, evidently crippling them permanently. Tables 3 and 4 show a number of the cases, and their outstanding symptoms.

Every case that I have reëxamined showed at least some residual symptoms. Case 4 illustrates this point very well. This patient was one of the mildest cases in the series, and yet showed practically all the symptoms that he had in the acute stage, with the exception of the myoclonus. Whether the other cases would show the same high percentage of residual symptoms, I cannot say definitely, but it may be safely surmised that the majority at least would show some symptoms either organic or psychic. All those who survive, it seems, are destined to bear some of the marks of this disease.

It is important to bear in mind that the severity or mildness of the condition during the acute stage of the illness has no definite relation to the sequelæ. The Commission of the Association for Research in Nervous and Mental Diseases (25), speaking on this point, says: "It must be remembered, in going over these papers, that the cases represented are practically all severe ones with marked symptoms of involvement of the central nervous system. Many of the milder forms, the Commission believes, never reached the hospitals, and many of these in all probability had no sequelæ of importance." This view is not borne out by experience. In case 4, if it were not for the erroneous diagnosis of nephrolithiasis, the patient would never have reached the hospital. After the pain subsided, he was an ambulatory case, and the diagnosis was made late in the disease, and yet at the reëxamination two years later he showed many residual symptoms. This is brought out even more strikingly in case 34, a definite Parkinsonian syndrome resulting in this case, that not only did not find its way into a hospital, but was only two days in bed and the diagnosis was not made until twenty months later when

a careful analysis of her history brought to light the true nature of her condition. It is true that the administration of an anesthetic and an operation precipitated the Parkinsonian syndrome, but the process must have been latent all during the fifteen months that elapsed between the mild acute illness and the resulting severe sequelæ.

Since I gathered the material for this paper, several other mild cases developing marked sequelæ, giving further evidence in support of this view, have come to my notice and will be reported at a later date.

In the consideration of these cases, I adhere again to the view that it is inadvisable to group them according to types, because the majority of them show mixed symptoms. In case 32, for instance, there is right hemiplegia, Parkinsonian syndrome of unilateral type on the left, and in addition, symptoms of myasthenia. In case 7, there is hemiplegia on right, evidence of dystonia musculorum in the right hand and arm, intention tremor on left, and in addition, fairly marked psychic changes.

A study of table 3 will show the residual symptoms encountered in cases that came up for reëxamination. A study of table 4 will show those of marked sequelæ. On account of not having had the opportunity to reëxamine all the cases observed during the acute stages of the disease, actual percentages of each group cannot be given, but the number of complete recovery must indeed be small, since all those reëxamined showed at least residual symptoms, if they were not permanently crippled.

The prognosis for ultimate and complete recovery from this condition, therefore, should be guarded.

GENERAL DISCUSSION

The question arises in my mind whether the name Epidemic Encephalitis is justified in view of the fact that we frequently encounter sporadic cases that are definitely encephalitic. It is a point worthy of consideration. These cases are still very puzzling to the profession in general and the diagnosis quite often is delayed for a considerable length of time. A case that I have at this late date, December, 1922, in the hospital, illustrates this point forcibly.

A woman of thirty-six, took sick late in October, 1922. She started with ordinary symptoms of infection and neuralgiform pains in the face. At first a diagnosis of pneumonia was made. A week later, a nose and throat specialist operated on her antrum and removed a simple cyst. Not until she developed semi-paresis of the entire left side of the body did the physician send her into the hospital

and the condition finally diagnosed correctly. She presents a typical case of encephalitis.

Again, taking into consideration the fact that many neurologists after encephalitis was established as an entity, could look back and diagnose retrospectively not a few cases that occurred before the epidemic, it would seem that encephalitis, like influenza, is always with us and that it lights up in certain years and becomes epidemic. In my opinion, it would be much better to designate the condition as encephalitis for what is known at present as epidemic (lethargic) encephalitis, and whenever there is another known factor involved, mention it, such as influenzal, toxic, or hemorrhagic encephalitis. Our experience in the next few years will prove or disprove the correctness of this view.

The question of contagiousness as well as the type of individual affected has agitated the minds of many observers. Lepine (26) thinks that there is always a predisposing factor such as excessive physical or emotional strain, or a constitutional nervous or mental taint. Netter thinks that the infection may develop in others in the patient's environment but that the disease manifests itself as encephalitis only when the nervous system is below par permanently or temporarily. Villiger (27) also thinks that the disease develops on the basis of a constitutional predisposition. That contagiousness is mild is agreed by most of the observers. We had at one time nine cases in a row in one ward at the Western Pennsylvania Hospital, no isolation methods were resorted to, and yet not a single case developed in the institution. That exhaustion and instability play their parts there is no doubt, but the exact mode of invasion or the reason why a certain individual becomes infected is not as yet known and remains a problem for the future to solve.

On the other hand, the type of reaction to the disease does seem to depend greatly on the individual affected, aside from the severity of the process. Thus, the two adults who had convulsions were both substandard types; one had a history of a period of confusion, or what might have been an epileptic equivalent attack; the other was of very poor stock, there was alcoholism in the family and he exhibited definite mental reduction. The same is true of the psychoneurotic nurse who developed marked hysteria late in the disease and had mild mental symptoms during the early stages. This probably also helps to explain the more serious sequelæ noted in children afflicted with the disease. Of the three children in this series, two remained permanently crippled, physically and mentally, and the third, while he could attend school, showed many residual symptoms,

easy fatigability, lethargy under exertion, and some changes in character. Paterson and Spence (28) report similar sad results in children, although they report six out of twenty-five cases as completely recovered. It would seem that the younger brain is not as resistant to the infection or that it has not the recuperative powers necessary to cope with it.

As to the fate of the post-encephalitic Parkinsonian, we have to change our erstwhile views. It was almost the universal belief that these cases tend to get well. Hunt (14), answering to a question as to what becomes of the encephalitic Parkinsonian, stated: "I think that the consensus of opinion is that this group of cases tends to improve." This is true with a large percentage of cases that exhibit the symptoms during the acute stage, but a fairly representative group of cases, especially those which show the symptoms some time after the acute illness, tend to progress and pass through the regular stages of paralysis agitans. I observed case 34 for a year and a half. At first only the left arm was involved, later the left leg became rigid and tremulous, and by now she complains of symptoms on the right side. Pette (29) reports a similar case of very mild encephalitis, which several months later developed severe Parkinsonian symptoms which persist. Evidently the disturbance, once it starts, tends to progress in certain individuals. There may, of course, be some inherent weakness in the striatum of these people which renders it more liable to undergo degeneration. This, however, can only be conjectured. A large amount of pathologic material would have to be studied before this point can be established.

The same question of chronicity and tendency to progression applies to all the other cases that showed marked sequelæ. It seems that a certain degree of permanent damage to nervous tissue has been done, which is beyond repair. The usefulness of these people seems to be permanently impaired. This is particularly true in those that show mental changes. The majority of them are depressed, lose all interest in life and are a burden to themselves and their family.

CONCLUSIONS

1. Thirty-five cases in all have been presented. Twenty-six acute, and nine retrospective cases.
2. The end-results have been studied in seventeen cases; seven have presented residual symptoms not interfering very much with the individual's usefulness; ten have marked sequelæ and are permanently incapacitated.
3. The percentage of complete recoveries cannot be given owing

to my inability to reëxamine all the cases, but all those who were reëxamined showed at least residual symptoms.

4. Certain symptoms that have not been stressed by other observers have occurred with sufficient frequency to deserve emphasis. Notably among them are: the frequency of altered reflexes and their persistency, rash and desquamation, the Babinski reflex, pains preceding myoclonic movements, hypotonia, reversed sleep, convulsions, and cephaloptosis in adults.

5. The presence of altered reflexes and pupillary changes, both of which are closely associated in our mind with cerebrospinal syphilis, in patients presenting themselves for examination, should make us think of encephalitis, and search carefully for it in the patient's history.

6. Post-encephalitic Parkinsonian conditions may progress and go through the same cycle as the true Parkinsonian. This is especially true in cases that have persisted for a longer time after the acute illness, or when the symptoms came on after a period of supposed recovery.

7. The mildness of the original attack bears no relation to the seriousness of the sequelæ. Many mild cases have developed serious sequelæ.

8. The administration of an anesthetic to a post-encephalitic patient should be avoided as long as possible. It may light up a latent disease focus.

CASE REPORTS

Case 1. Lethargy, mild Parkinsonian syndrome, fleeting paralysis, pyramidal tract involvement, desquamation, early delirium, and radicular pains. J. A. D. No. 3855. Admitted 11/1/19. Disch. 12/24/19. Age thirty-nine; American; white; foreman; married. Both parents died of pneumonia. Two brothers and two sisters living and well. Two brothers dead, cause unknown. Wife and six children living and well. Usual diseases of childhood. Typhoid sixteen years ago. Habits, good. Dizzy both in sitting and recumbent positions. Tired, worn out, pain in back of head which radiates down the neck.

The patient took sick two weeks before admission to the hospital. The first symptoms were tiredness, lack of vim and vigor, and restlessness at night. On the third day headache developed. On the fifth day diplopia came on and a slight rise in temperature, 99 F. On the seventh day the doctor noted paralysis and photophobia, requiring a darkened room. In a few days he became delirious, talking mainly about his shop work. On the twelfth day he became lethargic and had to be roused for his meals.

On the fourteenth day he was brought to the hospital, and the following notes were made on the first examination: The patient

has marked diplopia. Mask face marked. Facial folds obliterated. Pupils contracted, unequal, right larger than left, irregular, and do not react to light. Slight ptosis of the eyelids, particularly on right. Photophobia pronounced. Slight weakness of right face. Festinating movement of eyes. Slight paresis of both external recti. The patient protrudes his tongue slowly with slight deviation to right. There is a nasal twang to the voice. There is tremor of both hands. No hypotonia. No muscular rigidity. No adiadochokinesis. Left biceps and triceps reflexes increased. Abdominal reflexes diminished. Deep reflexes of left leg diminished. Right knee jerk normal, right achilles increased. Great toes do not respond to stimulation, but no Babinski. No ankle clonus. No sensory disturbances. No Kernig. Slight pass pointing on left.

The patient is disoriented at times. However, he knows that this is a hospital and that the doctor is examining him.

Scaling of face, arms and feet were noted on the next day. Two days after admission, perceptible weakness of the flexors of the left leg and increased patellar reflexes were noted.

11/4/19. Facial paresis on left more marked than on right—progressive paralysis. Pronounced atrophy of left leg. Suggestive Babinski on right. Face, especially around mouth, greasy.

11/7/19. Facial palsy cleared up. Desquamation of arms more marked. Babinski definitely positive on right and suggestive on left. Deep reflexes unequally increased.

11/10/19. For the first time since the first examination a fine regular tremor of hands is noted, also the cog-wheel phenomena in arms, especially left. Muscles of right leg beginning to be flabby. Diplopia absent, pass pointing present.

During the next two weeks, the paralytic symptoms were all regressing, but he complained constantly of severe pains in the head, neck, elbows, and hips. There was desquamation of the feet, especially the soles and around the toes. Strength in the left leg was returning gradually.

During the next month of the patient's stay in the hospital, the signs and symptoms would alternately regress and become aggravated. On leaving the institution he still presented a typical masked face, a practically stationary right pupil, increased deep reflexes, positive Babinski and Chaddock on right, and marked trophic changes on the soles.

The temperature was usually between 99 and 100 F. The pulse was in keeping with the temperature.

Case 2. Convulsions, choked disc, pyramidal tract involvement, confusion, and hyperidrosis. Age thirty-three, No. 4817. Admitted 12/9/19. Disch. 1/8/20. American; white; draftsman; single. Typhoid fever thirteen years ago. Denies lues and Neisserian infection. Is heavy smoker of cigarettes and pipe. Convulsions, headache.

Onset of disease about twelve days before admission with fever of 102 F., and sense of tightness around the head. He was sick about one week and had evidently commenced to improve, but in

about five days vomiting began. The vomiting was associated with nausea. This kept up for three days and during the night of 12/8/19 had the first convulsion. He had three convulsions at intervals of three hours. They were typical major attacks.

On admission to the hospital, the following note was made: The patient is confused. Thinks that he is in a suburb, and that he met the examiner daily in that town. He complained of sense of constriction around the head.

Pupils normal. Ocular movements normal. Fundi—right, practically choked disc; left, margins are hazy. Face somewhat mask-like. Tongue toxic, protrudes well, and shows evidence of sore—bitten during convulsion. Right knee jerk slightly increased. Exhaustible ankle clonus on left. No Babinski. Abdominal reflexes diminished on left.

12/12/19. The patient had a convulsion during the night, lasting about a half hour. He also had one the night before. He bit his lips severely. There is twitching of the labial muscles.

12/13/19. The patient's family physician relates that about eight weeks before, the patient had an attack of confusion. He lost his way even though he was in immediate and familiar surroundings.

The left pupil is sluggish to light. Nystagmoid twitch of left eye. Right fundus considerably cleared up, but margins are still hazy. Tongue slightly deviated to left. Deep reflexes on the left are increased. No Babinski, ankle clonus, or sensory disturbances.

He is particularly anxious that his mother should not be told about his sickness. (His mother died two months before.)

The ophthalmologist examined him the same day and reported "peripillary edema of right eye; left eye negative."

12/15/19. The deep reflexes have become increased.

12/28/19. Complains of marked sweating during the night. It keeps him from sleeping. The sweating was confined to the lower part of the body. He also complains of pain in the supraorbital region.

Temperature on admission 101 F. On the third day it was down to 99, and stayed around that for about two weeks. From then on, normal. Pulse around 90.

Reëxamination

3/23/22. The patient states that he has been a little nervous every now and then, that he would get a sensation of tightness around the head like a band. It would come on after eating.

Pupils slightly irregular, especially left. Both react sluggishly to light. Ocular movements normal. Fundi normal. Tongue deviated to right. No facial palsy. Slight tremor of right hand only. Deep reflexes of arms increased, slightly more on left. Deep reflexes of legs are increased, much more on right. Lower abdominal reflexes very much increased. Grip of hands good. No sensory changes.

He thinks that he does not have the same "pep" he used to have. Sleeps well. There are no mental changes.

(To be continued)

TRANSLATIONS

EMOTION, MORALITY, AND BRAIN*

BY PROF. C. V. MONAKOW

ZÜRICH

(Continued from page 68)

Every attack of this sort finds explanation in the ideational current of the patient, namely, that the patient usually clings to the most closely related fact or to related experiences of the remote past. The whole subjective causality straightway becomes mobilized with compulsion, to stress this once more; strange causal relations are invented and are firmly adhered to. The most false causality belongs necessarily to the attack! The patient is convinced that a localized bodily illness resides within him (the heart, digestive apparatus, sexual organs, or other organs are affected), or else "overwork" is cause of the anxiety. Later still other more remote motives, such as death or sickness of relatives, are drawn into the explanation, or bad air in the room at the time of the attack, digestive trouble, in short, all except only the "complex" itself. This misleading subjective causality may become later the basis for secondary symptoms. Many somatic and psychic troubles, even the search for an explanation fitting the attack, are fixated or arise periodically, generally as compulsions.

Thus phobia develops on the foundation just now sketched. Phobia is a condition in which negative illusions (fear of something unexpected, evil) exert their deceptive influence with regard to the past and the future, that is, retrospectively and prospectively carried on, under the restraint of correcting conceptions (true instinctive obscure thought, impelled by the "complex"), and all this in spite of the formerly existing logical sequence and intact nature of thought. Speedy release and care for the most important life interests, which follow his emotions in danger, form the focus for the thought of the patient. Eventually a disconsolate emotion takes possession of him, of having barely escaped an immediate, great danger or painful

* Authorized translation by Gertrude Barnes, A.B., and Smith Ely Jelliffe, M.D., of the authors *Gefühl, Gesittung und Gehirn*.

situation; in my opinion he becomes the victim of retrospective meditations: what immense evil could have come to pass, if the danger had not been removed in time (for instance, to have been crushed by driving), and what great danger may not await him? Such thoughts, that is, the inner retrospective examination of an imminent peril, and the possibility of a return of such in the near future, is always reproduced anew compulsively, and they conceive anew a fear of fear, unless some rescuing voice brings the true relationship of things into the ideational sphere of the patient, whereby at least a temporary discharge of the torturing tension is brought about.

Phobia constitutes a chronic latent condition of great lability. It is released periodically through mnemonic stimuli (symbols, allusions, and the like), and is characterized by short or protracted attacks. According to the innervation region in which the mnemonic stimuli is "projected" (periphery, instincts, causality), and according to the reaction on the part of the individual, we encounter differences in form, that is, differences in symptom complexes, which betray a mode of reaction in keeping with the form of reaction to which the individual is disposed.

Form 1. The immediate life (respiration, heart action, and so forth) appears in the eyes of the patient to be menaced, perhaps only under certain conditions will come to pass, if he undertakes this or that, or if he forbears doing this or that. He cannot set foot on a wide square, go alone on the street, go past certain houses, and so forth, else something undefined but evil may confront him (agoraphobia). In one case a patient is driven from within to leave a meeting hall filled with people, to flee; even the idea of visiting such a place without protection awakens anxiety within him. In another case, if he has touched a suspicious object he is tortured with the idea of having infected himself, of being the carrier of a dangerous disease (delusions of touch). The reaction in all is: away, flee, home, or wash, in short, a blind grasping for protective devices of all kinds. The patient plunges out, cannot be alone, the impulse leads again to perseveration (compulsive persistence), sound reflection is checked, crushed out (holding fast to the physician, to certain objects, and so on). In the latent state the patient avoids or goes around certain places, where a surprise attack, sexual violation, could menace him. His motto is: evade a possible danger or disagreeable situation at all costs (even fear of such a fear).

Form 2. The anxiety is in the same form as above, but the patient does not flee; he does not even resist the situation; on the

contrary, he remains passive; he remains silent and does not stir (inhibition). The conflict is active in the innermost circle of the instincts. Through the passivity (for instance, playing dead; even feigning death in animals), the individual seeks to guard himself from a "surprise attack," or he continues to cling to introspection. The patient, then, is held fast upon the spot where he happens to be; he cannot arrive at a resolution, cannot arouse himself energetically to his usual manual work, for instance, to dress or to undress. However, the visceral nervous system works at full pressure (tachypnea, tachycardia, expanded pupils, pale face, finally body excretions). This catatonic condition in schizophrenia, accompanied frequently by hallucinations or delusions, can give way in the various phobias after a few minutes, or after a longer time; there is a remission, and the attack threatens to return, until medical help is at hand.

Form 3. The character of the attack is changed (fear of the attack, which is postponed); it is a question of a latent, but continued labile condition with variations or remissions. Here, however, projection of the mnemonic stimuli (of the "complex") takes place in the "world of instinctive reflection," but with greater opposition of logic, because the affect is no longer in an acute state. The means of defense against the threatening danger (this lies more in the future, in the eyes of the patient, and is dependent on special contents of thought) constantly occupy the patient; the most unlikely and most remote motives are often involved in the explanation, and the "reason" apparently "descends from Heaven."

The instinctive, logically loose reflection over the suitability of the means of defense chosen is released with compulsory force, but here the patient is entirely conscious of the compulsion and of his foolish conjectures; but he is defenseless against them. One suffering from the *maladie de toucher*, that is, the fear of touching, cannot keep away from the washbasin; a hundred times a day, indeed, he must wash his hands. If he is prevented forcibly from doing so, or if he controls himself (and he can), then an acute anxiety attack with all its "visceral" consequences may break out. Others surpass themselves in devising means of safety (repeated looking about to see if someone is under the bed, whether the door is actually locked, seeking to save one's self by a sudden step), and so on.

Form 4. Again here it is a question of a labile latent state of anxiety, which bears intermittently the character of torture. Here the situation rules the "fear of fear." The projection of the abnor-

mal mnemonic stimulus (affectively toned experience in the remote past, which is held only darkly in the memory) takes place in logically more vigorously grounded ways in the sphere of subjective causality. In this form the patient falls victim to a real "metaphysical mania." Under continued compulsion the most various and apparently indifferent things (apparently furthest removed from the psychic trauma) are thought of as possible causes and effects. A true "causality delirium" or mania for questioning takes place (with painful care for all eventualities). The patient can indeed give a correct answer to himself; indeed, the answer appears to him as superfluous and as foolish as the question; but notwithstanding this he can never calm himself. Why has a chair four legs and not three? What would be the consequences if his healthy child had been born a monster? Why must we eat? And so on. Other questions apply to very definite delicate experiences, or have close analogy with them; thus it is perhaps a question of perils threatening the patient in his environment because of indiscreet assertions; or the questions pertain to inner psychic conflicts (of a nature important for preserving life, sexual, social, religious) with unsatisfactory or stored up discharge (feeling of sin and anxiety). One of my patients under compulsion had to blaspheme against God in thought to defend himself from an anxiety attack, and helped himself against this means of defense, which seemed absurd and unagreeable to himself, at times in equally foolish ways, although with temporary success, if he applied the respective blaspheming words in thought to all his acquaintances, so that God, the father, would be eliminated from the conflict.

Form 5. These are compulsory ideas or emotions based on certain definite experiences, and struggling towards a discharge through activity. They are deeply, affectively toned, and the latent anxiety is here conditioned or dependent on special stipulations; it does not devolve upon the immediate present, but refers to the past or the remote future: the patient might shriek out loud in church or other public locality a sexual secret very painful to him, or compromise himself in other ways, and so on. Or: in what evil condition might he not have fallen years ago if a small incident had not saved him at the last instant.

Here the typical feature is the compulsive causality, turning over monotonously on a possible conflict, and usually weak in its logical structure. The conflict, to accent this once more, is comparable to a convulsion in the motor sphere. And for removing the conflict are

chosen the most peculiar psychic means (charm, symbol, speech, and so on), according to the nature and education of the patient.

I will now leave the interesting subject of Phobia, which still needs much elucidation, and devote to-day a few words to Hysteria, that well known neurosis, in which the unreleased emotional conflicts, heaped one upon another, are brought into a latent state, and injure principally the "world of sensation and motion," that is, the world of ideas, and thereby, of course, they injure those corticosomatic functions (essentially temporal), which above all evoke active and inhibitive stimuli, whereby they produce symptoms on the part of the visceral nervous system similar to those in other neuroses. But here, also, this comes to pass without especially injuring the real "intellect" and thought. In hysteria it is a question of course of psychogenically produced phenomena of paralysis or excitement, through mediate or immediate influences on the life of the soul, which are often definitely bounded off specially, and may affect any part of the nervous system. These stimuli are easily demonstrable even objectively. They resemble organic disorders in manifold directions: paraplegia, monoplegia, hemiplegia, abasia, amourosis, hemianesthesia, and so on; and even severe disorders of the secretions—vomiting, diarrhoea—(secretion of saliva and perspiration) appear.

How can the severe "functional" disorders, where the "soul" ("psychic" forms of stimulation, "mnemic-irritative") interferes in a relatively rough, inhibiting, or irritating way with the tectonically delicately organized brain structure, be explained physiologically and biologically on the basis of our present day knowledge? We must remember in this connection that what we call "soul," biologically is a living structure within us, coming into existence out of the various biophysical and biochemical processes that have occurred in endless complicated developmental phases, and therefore taking the form of innumerable "temporal layers." The soul is a collective concept, which presents itself to its possessor in a form wholly different from its biological structure. For this form we have as yet found no appropriate method of physiological investigation or approach. In my opinion the psyche is comprehensible only "historically," that is, genetically, and only as a continued series of complicated reciprocal actions (even various reciprocal "inhibitions") between the outer world and the world within us, and when the inner secretions are duly taken into consideration.

In organic paralysis (for instance, hemiplegia) a severing of

continuity in the great motor area (inner capsule, tegmentum, pyramids) is assumed as a direct anatomical fact. The initial flaccid hemiplegia constitutes not only the result of a severing of continuity anatomically; it presents rather a combination between the indispensable necessary motor results of a lesion of the corticospinal path (residual hemiplegia) and of a functionally caused false distribution of innumerable other impulses participating in the construction of the bodily movements: dynamic components. The anatomical basis for this is composed of structures widely separated from each other but connected with the lesion by means of medullary fibers. A process here seems to take place which presents resemblance to shock and which I have called "diaschisis" (corticospinal and associative diaschisis).

In this connection, injury to the circulation in various cortical and other areas, and also the pathological process, etc., play an important part. The gross anatomical components, the "skeleton" of the residual hemiplegic symptom complexes, especially the typical spinal symptoms of disintegration (change in the various reflexes of the extremities: the Babinski phenomena of the toes, the "cumulative retraction reflex," and various other synkinesias, etc.), are missing almost without exception in hysterical hemiplegia, while many other initial-hemiplegic symptoms (hypotonia with limp, dragging of the leg, significant motor weakness, and so on) stand impressively in the foreground.

For the elucidation of the pathological neuromechanics in hysterical hemiplegia, we must consider primarily the innervation weakness, and then the dynamic moment (false distribution of the corticospinal impulse), since gross anatomical disintegration of the motor components is missing. It is a dynamic disorder, which is closely related to a diaschisis, but for which other anatomical points of attack must serve as a basis entirely. Here not only the circulation of the blood but also the injurious influence of certain chemical combinations in the blood may play a rôle: both moments just now mentioned—we cannot avoid this conclusion—must exert an unfavorable influence on well-defined anatomical connections of the central nervous system (projection and association components) from a certain level of cerebrospinal tectonic anatomic development on. Very definite cellular elements and structures must become refractory for the spontaneous (intracortical) stimulation. Or else certain levels in the cortex may be unstimulated, but which enter upon the scene before spontaneous motor impulses; levels, that is to say, which serve as the

basis for the successive development of unilateral movement, the exact localization of which cannot be more definitely determined at the present time. These are apparatuses distributed according to historical development and built up according to definite temporal principles (inhibition of the motor impulses involved).

If we adhere to the picture of a faulty distribution of the will impulse and relative "instinct stimuli" in the cortex, we might then conceive that through the psychically produced collision of the various forms of stimuli, on the one hand, an excessive burden with latent forms of stimuli in the sphere of the life of the instincts (a chemical form of diaschisis?; defense reaction, conflict on the part of self-preservation against foreign intruders), and on the other hand, excessive inhibition up to total immobility in the successive, rhythmically ordered development of the muscular mobilization, takes place. The last might perhaps be conceivable in the form of an overthrow of the successive rhythmically built up discharge: a transformation of acts otherwise following one another into a simultaneous discharge produced by summation or perhaps in such a way that the natural tectonic-anatomical points of attack for the stimuli would no longer respond. The further phenomena in relation to the innervation of the extremities then would be of secondary nature (results of faulty regulation), and they would be of a very similar kind as in cerebrospinal diaschisis. In other words, even in the secretions, a loosening of contact along the whole line, even to spinal reflexes, would break out, a loosening of contact which nevertheless leaves a certain room for combined cortical and subcortical reflexes to play. In morphological and general physiological sense, one might incline to assume interruptions between the stations for the mnemonic motor impulse and the cortical innervation points, for the realization of the movements (cortico-associative system of electrically excitable foci). Herein an involvement of the chemism regulated through the inner secretion (even in the cerebrospinal fluid?) would not be excluded, although this would not explain the one-sided innervation injury nor the localization.

This question arises therefore: in hysteria—where it is a question of an injury to the total innervation—why, as soon as the bodily parts are concerned, are only *very definite*, spatially outlined bodily segments and sensory spheres chosen, and why do all the rest remain free? How does the election with regard to certain peripherally sharply outlined innervation spheres take place as result of a general injury? In one individual we find, as is well known, amauroses as

the result of a psychic trauma; in another, hemianesthesia; in a third, paraplegia; in a fourth, ischuria or mutism, and so on. One usually connects these elections with the special content of the single or several "complexes," with definite conscious or unconscious "over-determined" ideas or images, and seeks to explain even the somatically localized hysterical symptoms on purely psychological grounds. But in my opinion the various somatically localized hysterical symptoms are much too gross for such a purely psychological explanation ("autosuggestion") to be satisfying. In many of the inhibition phenomena in question (hysterical paralysis), even biophysical and biochemical functioning moments must be called into play in parts of the cortex marked out in a rough way (perhaps corresponding to great projection fields or arterial areas in the cortex). These moments we can designate provisionally in no way except as "psychic shocks," produced by summation. This may be separated from the more anatomically considered diaschisis (separation according to fibrillar components in the surface of the fracture) only in the sense that in the latter case, the points of attack or injury are defined through *anatomical* course in the foci of interrupted bundles and fibers; in psychic shock, on the contrary, they usually spring from components of stimulation usually built up according to a temporal plan and not sharply differentiated anatomically (injury in paths of innervation elaborated and adapted physiologically and biologically).

One might think of other apparatuses involved in this choice—aside from the autotoxic and vasomotor components—apparatuses which already at the time of illness had been organically weakened. Or one might consider the stations of innervation which were less resistant in consequence of deficient exercise and training.

Here we reach a new point which is of cardinal importance, especially for the comprehension of spastic and paralyzing hysterical conditions, namely, the problem concerning the importance of the struggle for vital interests in question, concerning the arrangement of the various "innervation layers" which, as they do not admit of being more closely determined spatially, must be considered more chronologically, in the light of the genetic succession of the various nervous performances. Here we must proceed from the view that in every conflict concerning the importance of a physiological act or activity, that is, its "birth," the central apparatus serving for the preservation of immediate life, then those serving for the "defense" of the separate bodily parts and organs, other things being equal, are not

only ontogenetically and phylogenetically oldest, but they are by nature by far the best adapted to resist external insults. These apparatuses are: circulation, respiration and the defensive reflexes as well as those for preserving equilibrium of the body. The apparatus for orientation, sensory perception, for preserving the upright position, and indeed for the intended movements as well as for gestures are acquired much later, and are scattered. Notwithstanding several well defined cortical "centers," they are as far as their functioning is concerned, assigned to innervation acts, which are very complicated in their temporal construction, and dependent on reciprocal influences. At any rate their function may be destroyed from many directions, and in a relatively easy manner, both directly and indirectly (sopor, coma, stupor). They may be injured more easily and more profoundly than the primal apparatuses. This to a much greater degree is true for those innervation spheres (fibrillar bands, molecular substance, the satellite and the granular cells), which in the child are built up later and more delicately, and the activities of which necessitate a well organized and constantly more delicate relationship between the various phases of psychic development. They remain capable of performances only if the psychic historically developed structure (succession continuity of the important personal experiences, that is, the temporal orientation and the results proceeding from them) is not essentially disturbed, and if the inner secretions function correctly.

Within these last processes and forms of stimulation, there are found both in the intellectual sphere and in that of morality, activities of a still higher character (religious, artistic endeavors and needs, virtues, duties, and so on), which win the victory in the competitive struggle with inferior motives and instincts, and can continue in their hard-earned higher level, only if they are continually exercised, and constantly strengthened through new experience—often at the expense of the lower instincts. These highest psychic activities, now, are especially easily injured through acute psychic shocks or experiences of many kinds, and also through pathological (infectious) processes in the cortex, as well as through endogenous and exogenous poisons and they are the more easily injured in proportion as they belong to later developmental periods or levels in the child and in proportion as they presuppose more complicated physiological conditions as their foundation. Often failure or difficulty in functioning in this or that nervous, or psychic sphere has been dependent not in small measure upon the order of the morphological and biological

developments of the apparatus in question. Moreover, individual tendency, discipline, and education, and, too, the significance of the activity for the life and success of the individual as a whole, play an important rôle. And in my opinion, combined mnemonic factors of stimulation, similar to those built up through the individual development and education—together with their interest values—deposited in temporal strata (among others, those brought together for us step by step through school) can disappear also “in layers” or step by step—or indeed becoming inhibited, as it were, or disintegrated, according to the “educational phases.”

We come now to the question: How can we explain that in phobia and also in hysteria, the insults, for example in the sphere of sexual life, produce *bodily symptoms* (among others, in nervous attacks) in *entirely other visceral* innervation spheres, that is, not within the injured sexual functions, but within the uninjured functions important for life; thus for instance, cardiac, respiratory, digestive symptoms (abnormal heart action, feelings of anxiety, difficulty in swallowing, dyspepsia)? Does the process of displacement always follow certain (perhaps affect toned) ideas, certain symbols, or is the way determined rather by physiological (organization of the visceral innervation) or even anatomical causes (tectonic structure, vascular supply)? From a wholly physiological viewpoint one would think, that for instance the sudden, disagreeable discovery of the impotence of the husband, or of his faithlessness would produce in the wife sensations and other difficulties within the sexual sphere (for instance, vaginal spasms, ovarian and sacral pains, and so on) but not an arrhythmic heart, tachypnea vomiting, feelings of anxiety, vasomotor disturbances, and so on; although in concrete cases it might not be difficult to find a connection between the trauma and the bodily disturbances by way of the play of thought and symbols; and this without proceeding arbitrarily in the interpretation.

(To be continued)

SOCIETY PROCEEDINGS

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

REGULAR MEETING, OCTOBER 18, 1923

F. K. HALLOCK, M.D., President, in the Chair

A CASE OF MYELITIS OF UNUSUAL ETIOLOGY

DR. J. W. COURTNEY

The patient I have to present is a man, aged forty-seven, and married. He was born in Ireland and has been in America 28 years. For 25 of these years he has worked as a club servant. He was referred to me August 17 of the current year, by Dr. W. R. MacAusland.

The family history is unimportant. His mother is alive at ninety. His father died at an advanced age, cause unknown. He has four brothers and five sisters, all well. There are no nervous or mental diseases in the family. Previous history: The patient was married at twenty-five years. He denies venereal disease absolutely. He has not abused alcohol. His health was excellent up to October, 1922, when he had an exceptionally severe attack of bleeding piles. For two months the loss of blood at stool was profuse. At the end of that time there was noticed numbness of the right great toe followed by a burning sensation in the ankle. This was followed by a trepidation of marked degree in the muscles of the right lower extremity. The numbness gradually extended from ankle to knee and from knee to the lower abdomen. About three months later numbness and trepidation took possession of the left lower extremity. The numbness ultimately reached a level just above the symphysis. At that time great difficulty was experienced in starting the urinary stream, although the urine was passed freely with the stools and the patient could feel the flow. Defecation was very difficult, but there was no loss of sensibility in the rectum. There was no fever and the patient was not confined to bed. There has been no pain whatsoever aside from what the patient describes as a "neuralgic feeling" in the knees. For eight weeks prior to my first examination there was dragging of the right leg, and for three weeks the left leg had been spastic. There has never been any disturbance of the power of erection. The upper extremities were never affected. The numbness experienced up to the time the left leg became spastic was purely subjective.

Physical Examination: The patient was a fairly well nourished man but rather pallid. He assured me that he had never had much

color in his face. Progression without assistance was impossible. The gait was not only spastic-ataxic but it was further embarrassed by an extraordinarily severe myoclonus which involved practically all the leg muscles. The cranial nerves were normal in every way. The upper extremities were likewise normal. The heart area and sounds were normal. The spine was normal in contour and flexibility. With the patient seated, the clonic spasms were markedly diminished, but any voluntary movement increased them at once. Muscular atrophy was entirely absent. All forms of sensibility were blunted but not lost throughout the legs to the level of the symphysis. The knee-jerks and tendo-Achilles reflexes were enormously exaggerated. The slightest upward pressure on the plantar surface of the foot produced a violent and persistent ankle clonus. The Babinski phenomenon could almost be produced through the sole of the shoe. Weakness of the lower extremities was marked. In arriving at an etiologic solution of the case I could not escape the conviction that the appearance of the primary numbness of the right great toe synchronously with the severe hemorrhoidal attack was not to be regarded as a mere coincidence. The free anastomosis of the hemorrhoidal system with the spinal venous system suggested very strongly that one of two things had taken place: either an upward extension of hemorrhoidal thrombi or an infection through these veins.

With this idea in mind, I returned the patient to Dr. MacAusland and urged an immediate exploration of the rectum. This exploration was made by Dr. Ralph Jackson, who is present and will, I hope, tell us what he found and what he subsequently did in an operative way. The trepidation practically ceased before the patient left the hospital after Dr. Jackson's operation. Since his discharge from the hospital, his locomotive powers have improved to such an extent that he now makes his way about the house without any artificial aid whatsoever, and gets along the street with the aid of a cane alone. The spasticity is still distinctly present, and more marked in the left leg than in the right. The ankle-clonus and the Babinski sign still persist, but the patient has been gaining constantly in flesh and strength. I am rather inclined to believe, in the last analysis, that the cord changes, which must have found their highest level opposite the fourth sacral roots, are nutritional and that they were brought about by blocking of the venous outlet at that point.

Discussion: Dr. Ralph W. Jackson: We have a rather unusual condition in this patient's rectum. Dr. Courtney was very positive that there was some rectal pathology. I found a very definite internal hemorrhoidal condition, more marked on the left side of the anus, and an indurated column running upward toward the seminal vesicle on the right side of the ampulla. This latter was not tender or discernably fluctuant, felt like scar tissue, and was suggestive of a sinus, though I could find no opening. We decided on operation, and I first incised the indurated column, which collapsed somewhat, and may have contained a few drops of pus, but if so, it was not discernible on account of the blood from the hemorrhoids. I then

did a radical operation for the hemorrhoids, enucleating them in a manner that is as different from an ordinary hemorrhoidal operation as a tonsillectomy is from a tonsillotomy. The hemorrhoids were not excessively large nor more definitely infected than in the ordinary case.

The rectum and anus, as possible foci of infection, are sadly overlooked. I have seen a number of cases of arthritic conditions which have been benefited or cured by adequate drainage of obscure fistulous tracts therein, and the possibility of absorption of infection from infected hemorrhoids is undeniable. I have had no experience with distant consequences arising, as Dr. Courtney has worked it out, from venous thromboses originating in the ano-rectal field, but anatomically there would seem to be no reason to deny the possibility of such conclusions; and they would seem to be justified in this case in view of the results of operation, which were so prompt and astonishing, that I thought at first they must be psychological, but they are continuing and the patient is getting well.

Dr. F. K. Hallock: Has anyone else had a similar experience? Perhaps the rectum will come to be as important as the teeth and tonsils.

Dr. Courtney: I would like to say a word about another case seen by me some years ago with Dr. Nathaniel Hunting, at the Quincy Hospital. Dr. Ayer later made a necropsy on the patient. The latter was a strongly built Swede. Some weeks before I saw him he was assisting some other men in unloading a hen-coop from a dray. The coop slipped and produced an abrasion of the skin of the patient's right chest wall anteriorly. Within a week a small abscess formed at the site of the abrasion. The physician to whom the man went paid no special attention to this abscess. Within a relatively short time the patient noticed an increasing embarrassment in his locomotion. With this there were later associated anesthesia and vesical sphincter disturbance. When I saw the man he had all the signs of a transverse myelitis. My opinion was that the pus had traveled back through the lymphatics to the cord. Dr. Ayer's necropsy findings confirmed my diagnosis.

Dr. J. B. Ayer: The case referred to was one of frank epidural abscess, which extended to the back from an original focus in the breast. The spinal cord at the level of compression was liquefied, due as we thought to the combined effect of pressure and toxic substances. Another case of epidural abscess was seen with Dr. Taylor, and although the pus was drained within 48 hours of the initial symptoms of myelitis, degeneration evidently took place, as the patient became paraplegic. We can hardly correlate these two cases of epidural abscess with Dr. Courtney's case. Such a mechanism as he works out in this case seems very possible in view of what we know of ascending toxic neuritis.

Dr. Courtney: Just a word in answer to Dr. Ayer. I see only analogy not identity in the two cases. I merely instanced the second case to show from what apparently innocent causes and through what roundabout routes the cord may be involved.

A CASE OF AMYOTROPHIC LATERAL SCLEROSIS SHOWING SIGNS OF PAGET'S DISEASE

DR. HENRY R. VIETS

The case to be reported seems to be unusual because a patient, with amyotrophic lateral sclerosis, also showed evidence of Paget's diseases of the bones by x-ray. So far as I have been able to ascertain, no case of this character has previously been reported in the literature.

The patient was seen in August, 1923, complaining of weakness of his voice, arms, and legs. He gave up work as a railroad executive in Pennsylvania three years before and had been spending his summers in a large gladioli garden in Massachusetts doing light work. He had marked difficulty in tying up flowers about one year ago. He passed, however, an insurance examination at this time, increasing his policy. His weakness slowly increased. In February of this year he became dizzy and fell in a railroad station in New York. There was no fracture and he left the hospital, where he had been taken, in two days. He has fallen twice since then. Shortly after this he noticed that he had difficulty in controlling his emotions; he would rather "overdo the part" and laugh or cry longer and louder than was consistent with the occasion. There were occasional choking spells and swallowing was slightly affected. Except for slight discomfort in the right shoulder, there was no pain. The voice gradually became deeper and of a nasal quality, growing weak during the day and giving out during the evening. He found difficulty in feeding himself and his gait became stiff and awkward.

Examination showed some moderate general weakness and slight bowing of the legs. His back was straight and there was no enlargement of the head. The pupils and cranial nerves were normal. There was slight if any atrophy of the tongue, but a rather coarse tremor was present. All the intrinsic muscles of the hands were atrophic with a corresponding loss of strength. These muscles showed the reaction of degeneration. The arm reflexes were pathologically increased and about equal. Numbness of the finger tips was complained of but no objective signs to prick, touch, hot or cold were found. The blood pressure was 140/70 without evidence of peripheral sclerosis. The sphincters were not involved. The legs were mildly spastic with an equal increase in the reflexes including a bilateral clonus and Babinski sign.

Lumbar puncture: 10 c.c. clear, colorless fluid. Pressure, 160 mm., pulsations and jugular compression within normal limits. Cells, 1. Protein, 31 mgm. per 100 c.c. Wassermann, negative. Blood examination: Wassermann, negative. W.b.c., 6,800. Hgb., 65 per cent. R.b.c., 4040000. Smear: polys., 62 per cent; lymphos., 30 per cent; 1 mono., 4 per cent; eos., 1; baso., 1. R.b.c. show moderate variation in size but none in shape; no achromia, basophilia or stippling.

Summary: A man of fifty-three has had for several years a slowly progressive atrophic paralysis of his hands and weakness of his voice. Associated with this is a mild spasticity of his legs and bulbar symptoms. The diagnosis first made was amyotrophic lateral sclerosis. There were no changes in the blood other than a slight secondary anemia and the spinal fluid was negative. Because of mild pain in the shoulders, bursitis was suspected and x-rays were made of this region. The appearance of the head of the humerus suggested Paget's disease and the pictures of the other bones confirmed the diagnosis as shown in the following report:

X-ray examination: The plates show a distinct variation from normal in the upper end of the humerus, the scapula, skull, pelvis, spine, tibiae, and femora. The bones are enlarged and somewhat irregular in outline with coarse irregular trabeculations and narrowing of the medullary canal in the long bones. The appearance was typical of Paget's disease.

Amyotrophic lateral sclerosis was first described by Charcot and Joffroy (*Arch. de physiol. norm. et path.*, Par. 1869, II, 354, 629, 744). Gordon Holmes in 1909 (*Rev. of neurol. and psychi.*, 1909, VII, 693) did some careful work on ten postmortem cases and laid special emphasis on the fact that the lesion from the pathological point of view was diffuse and not confined to the lateral columns and the anterior horns. Paget's original description and pictures of osteitis deformans (*Medico-Chir. Trans.*, Lond., 1877, LX, 37-64, pl. 5) are classical. Morton Prince (*Tr. Asso. of Am. Phy.*, 1902, XVII, 382) became interested in Paget's disease and investigated some cases to see if there might be some changes in the nervous system associated with the osteitis. He was not able to demonstrate definite lesions and very few changes have been found since that time, 20 years ago. One or two cases of Paget's disease, however, have been described with posterior column sclerosis, and one of chronic myelitis. Marie and Leri (*Bull. et mem. Soc. Med. d. Hop. de Par.*, 1919, XLIII, 904) have published a case of Paget's disease in which at autopsy they found syringomyelia. There was a large cavity from the first lumbar to the second dorsal segment. They had not suspected this finding and had not examined carefully for loss of hot and cold sensation in the extremities; certainly there were no gross changes of which the patient complained. It is interesting to note also that in Charcot's second case there seems to be a definite cavity of syringomyelia which shows in one of his drawings.

My single case then would simply add to the literature a case which seems to be definitely amyotrophic lateral sclerosis, and from the x-ray standpoint is Paget's disease. I was not able to demonstrate clinically Paget's disease in this man, although when looked over carefully after the x-ray examination, there was to be noted slight bowing of the legs and some of the arms, but no definite deformity of the skull. The plates of the long bones were remarkable, showing the well-marked changes associated with Paget's disease.

Discussion: Dr. E. W. Taylor: Dr. Viets was good enough to ask me to see this man in consultation, and I should entirely agree with his diagnosis. He certainly had a perfectly typical picture of what we regard as amyotrophic lateral sclerosis: atrophy of the hands and spasticity of the lower extremities and the arms. The question of the association of this with Paget's disease as merely incidental, or as a result of Paget's disease, is naturally a matter of great interest. It is hard to see how changes in the bones could produce this condition except perhaps by pressure on the cord. Of course there are sometimes changes in the vertebræ as well as in the long bones, and exostoses might occur and lead to pressure in the cervical region. I have no doubt Dr. Viets has studied the x-ray from this point of view. The x-rays are most interesting. The picture of Paget's disease is very pronounced though the patient had very few symptoms in spite of the changes in the bones. The whole subject of Paget's disease we cannot go into, for there are many conditions allied with Paget's disease: hyperstosis cranii, etc., and a large number of conditions ordinarily differentiated from Paget's disease have possibly a common etiology. It offers a large subject for investigation. The changes about the cranium and skull in this case are noteworthy, and it is possible they help to account for the conditions in the tongue and in the spinal cord which proclaimed themselves as a typical amyotrophic lateral sclerosis.

Dr. Viets: Typical Paget's disease will show an absorption of bone and a laying down of new bone of an irregular character; the increased calcification of the new bone is what you see in the x-ray pictures, combined with rarefied areas. The entire pathology I think is not definitely known. Just why these changes take place, or what they are, I do not know, other than the fact that abnormal bone is formed with a certain amount of absorption.

With regard to Dr. Taylor's point that the vertebral column might have affected the spinal segments, it is true that the vertebræ showed changes typical of Paget's disease, but there appeared to be no more changes in the seventh cervical and first dorsal vertebræ than in the other part of the spinal column. Pressure was ruled out by the procedures carried out at the time of the lumbar puncture. There was no evidence of subarachnoid block. There appeared to be some intrinsic disease of the cord.

That the changes may be neurogenic has been the theory advanced by Dr. Prince and many others and is based somewhat on the analogy of changes found in syringomyelia and tabes dorsalis, but many cases of Paget's disease have been analyzed since Dr. Prince's work and very careful work done in the last decade on the pathology of the nervous system without confirmation of the neurogenic theory. The case reported by Marie and the one described here might offer some evidence substantiating a theory of neurogenic origin.

OBSERVATIONS IN VENTRICLE AND CISTERN PUNCTURES

DR. A. H. RUGGLES

For the past two and one-half years, we have been interested at Butler Hospital in cistern puncture and its application as an avenue of approach in the treatment of paresis, as described by Ayer before this society. We have been impressed by the simplicity of the technic, by the lack of discomfort to the patient, and by the theoretical advantages of the more direct approach to the seat of the disorder. In applying intracistern treatment in general paresis, we have directed attention, as have a number of other workers, to the question of the passage of the cerebrospinal fluid from above downward and the possibility of passing fluids from the cistern into the ventricles. Solomon and his coworkers have investigated this point and published their results,* so that we are not offering this as original work—although at the time we did it we were not aware of Solomon's work—but rather offer certain observations which we have made, in most part confirming results of other workers, and also to extend our study of the best methods for the treatment of cerebrospinal lues.

Case 1. F. H. Age forty. Married. Admitted May 1, 1922. Diag.: General paresis. Duration: Two years.

April 14, 1923—Using local anesthesia (Novocaine $\frac{1}{4}$ -1 per cent) an opening was made in the right parietal region by means of Hudson burrs; after this a needle was inserted into the ventricle and a cistern puncture done, without draining the cistern, and 20 c.c. of a weak phenolsulphonphthalein solution injected to see if it could be made to flow upward into the ventricle. The ventricular fluid was allowed to drain off but after 15 minutes none of the above solution was recovered; then 20 c.c. of saline was allowed to run into the ventricle by gravity and this seemed slightly to increase the flow from the cistern needle. Nearly all of the phenolsulphonphthalein was recovered from the cistern. The wound was closed in the usual manner and the patient returned to the ward in fairly good condition.

April 20, 1923—In the first 24 hours after operation, patient's temperature went up to 104° , but with no corresponding increase in the pulse rate. The pulse was weak, however, and the night of the operation he was given 400 c.c. of saline intravenously and an ampoule of digifolin. The quality of the pulse improved and during the next 72 hours his temperature fell to normal; his pulse rate became normal and of good quality. He is now in approximately the same condition as before the operation.

April 23, 1923—Cistern puncture was done this morning and about 20 c.c. of fluid removed. It was examined for phenolsulphonphthalein and none was found.

* A. M. O. Jour., Vol. LXXIX: 1014, 1922.

April 30, 1923—This morning ventricular puncture was performed and about 5 c.c. of fluid removed and the needle left in place. A cistern puncture was then performed. A gravity cylinder, containing 20 c.c. of saline to which had been added 1 c.c. of a neutral phenolsulphonphthalein solution (prepared after Dr. Dandy's formula by H. W. & D.) was connected with the ventricular needle and 10 c.c. allowed to run in. The stylet was removed from the cistern needle and two minutes later the dye was recovered in the cistern fluid in very noticeable concentration; about 20 c.c. was removed by the cistern needle and then the cistern needle was withdrawn. A lumbar puncture was then performed and 10 c.c. more of the original solution was allowed to run into the ventricle. In twelve minutes it appeared in the lumbar fluid in very dilute quantities. About 15 c.c. of spinal fluid was recovered by the lumbar needle. It is possible that the dye would have been recovered earlier and in greater concentration had not the cistern been so completely drained. The patient was in good condition and complained of no discomfort after this procedure.

May 2, 1923—Ventricular puncture was performed this A.M. and slightly blood-tinged fluid was obtained. Cistern puncture was then done and slightly blood-tinged fluid was obtained here also. Twenty c.c. of a solution of phenolsulphonphthalein was allowed to run into the cistern by gravity (1 c.c. to 20 c.c. saline). The head of the table was lowered, the stylet was removed from the ventricular needle and in 50 seconds the dye was obtained in such concentration that it was very noticeable, even with the blood-tinged fluid. The cistern needle was removed. About 25 c.c. of fluid was taken from the ventricle all of which was strongly colored with the dye. The needle was then removed from the ventricle. Lumbar puncture was done and bloody fluid was obtained under very slight pressure. This pressure was increased when the foot of the table was lowered. Almost immediately the fluid became dye stained.

May 5, 1923—This morning lumbar puncture was done and a straw colored fluid was obtained.

May 7, 1923—This morning lumbar puncture was performed and about 60 c.c. of straw colored fluid was removed, which, however, was not so deeply colored as that obtained at the last puncture; the addition of sodium hydroxide shows no color which would suggest that any of the dye-stuff still remains in his subarachnoid space.

May 9, 1923—This morning cistern puncture was performed and 5 c.c. of dye-stuff was allowed to run into the cistern by gravity, but as no more would run in by gravity, 15 c.c. more were forced in by syringe. There was no change of pulse or respiration during the procedure. The head of the table was lowered. Twenty-five minutes afterwards the ventricle was punctured and about 4 c.c. of clear fluid was obtained. On draining the ventricle, dye-stuff was recovered 34 minutes after its injection and was obtained in considerable quantity. Lumbar puncture was performed and dye-stuff was obtained immediately in the fluid there but the pressure was very low—probably on account of the removal of ventricular fluid. It is our opinion

that while dye-stuff will flow from the cistern to the ventricle, when the ventricle is drained, no substance can be got into the ventricles of the brain without drainage of the lateral ventricle.

Case 2. E. A. K. Age forty-five; single. Admitted June 15, 1923. Diagnosis: General paresis. Duration: Eight months.

September 12, 1923—Yesterday patient was operated on under ether anesthesia and an opening made in the occipital region of the skull according to the route suggested by Dandy. It was found that to enter the ventricle, the needle needed to be directed downward almost toward the point of the ear. In this direction the ventricle was entered without any particular difficulty. Seventeen c.c. of serum, prepared according to the Swift-Ellis method, to which had been added 1 c.c. of neutral phenolsulphonphthalein, was injected into the ventricle. The opening was closed. Cistern puncture was then performed and a few drops of colored fluid obtained and the needle immediately removed. This shows fairly definitely that fluid injected into the ventricle will immediately pass to all parts of the ventricular system. Patient this morning shows no untoward or alarming symptoms, temperature, pulse and respiration being normal.

October 16, 1923—Since the time of the last note, patient has had nine ventricular punctures. Practically no difficulty has been experienced in entering the ventricle and the patient has suffered from no untoward symptoms following treatment. He has been a great deal quieter, showing a gradual lessening of activity since the time of operation. He sleeps fairly well at night; his appetite is good and he has had a gradual return of strength since the operation. His memory is better. He shows some flight of ideas, but this is not nearly so marked as previously.

In the first case reported, we were much impressed with the difficulty of passing fluid from the cistern into the ventricle, unless the ventricle puncture was made and the pressure in the ventricle first lowered, when it was possible to obtain the dye-stuff put into the cistern from the ventricle in a comparatively short time; but without first lowering the pressure in the ventricle, this flow was very slow and very limited. It is our belief, therefore, that intracistern therapy will not be conveyed directly to the ventricle except in very limited amount, and that treatment given into the ventricle very quickly circulates to the cistern below. Another point that we have lately determined is the ease of approach to the lateral ventricle by the method of Dandy as reviewed in *Surgery, Gynecology and Obstetrics*, May 1923.

In the second case reported we have already given nine ventricular treatments through the occipital opening and have had, much to our surprise, improvement in the case in which this treatment was employed, although such improvement was hardly expected as he had previously had prolonged intracistern treatment without any noticeable change. In approximately two hundred cistern punctures done since those reported by McCusker at Butler Hospital, we have had no untoward results; there has been less discomfort to the patients than

in a like number of lumbar punctures, and we have an avenue of approach more closely associated with the seat of disorder than is the lumbar region.

In those cases in which it is deemed advisable to give ventricular treatment, we would suggest, as the result of our limited experience, the occipital route of Dandy as offering no greater difficulty of technic and possibly greater ease in entering the ventricles. Our whole effort in the treatment of paresis has been, so far as possible, to study all the methods rather intensively, perfecting our technic so that in patients resistant to one type of treatment we might have others to employ and continuing treatment, in most of our cases, over a long period by the various methods as indicated. We hope to pursue our search until such time as we are satisfied that treatment of general paresis with the arsenic preparations in unavailing after all methods have been employed; at the present time we are encouraged with the results in our cases rather than the contrary.

Discussion: Dr. A. T. Wyatt: From studies I have made, I have found the anterior horn was very small from above downward, and immediately underneath the surface are the basal ganglions, which are extremely likely to be punctured. The posterior horn is not always present; in some cases it is absent entirely. It would seem from the sections studied that the best way of getting to the ventricle would be from a lateral position one inch above the orbital level and two-thirds of the distance from a line drawn through the midline posteriorly, and one through the external auditory meatus. The cistern is easier of access than the ventricle. The patients suffer less with headache from cistern puncture than from lumbar puncture, and do not complain of as much pain at the time of puncture. This method is not new; it has been done by Dr. Harry Cushing. I merely emphasize the advisability of going in from that direction.

Dr. J. B. Ayer: I have seen this method of ventricle puncture used both by Dr. Cushing and Dr. Mixter, and I think by Dr. Frazier. This work of Dr. Ruggles is of great interest. His results are in line with those of Solomon who finds a less concentrated fluid in the ventricles compared with that in the subarachnoid spaces, and with those of Dandy who experimentally shows that most of the fluid is produced in the ventricles, and by recent studies of Weigeldt who finds the pressure in the ventricles a little higher than in the surrounding subarachnoid space. We have then considerable evidence that serum does not easily enter the ventricles from subarachnoid injections, and on the other hand that serum administered directly into the ventricles may be promptly beneficial. The conclusions appear obvious.

Dr. Solomon: We found, a little differently from Dr. Ruggles, namely, that there was a little upward movement of the dye. The downward was more marked, but there was some upward current. I am interested in Dr. Ayer's difference between the ventricle and cistern pressures. We have made a number of experiments of similar type, taking the lumbar and ventricular pressures simul-

taneously, and found they were identical in many instances as nearly as we could tell. If we moved the patient so his head was a bit lower, the ventricular pressure would rise and the lumbar fall, nearly simultaneously. So with the withdrawal of fluid from one place or the other, the difference in pressure is nearly simultaneous as nearly as we could determine.

As to where to go in for punctures: it is a very simple procedure wherever you go in. We have used the frontal, going into the anterior horn, where the ventricles are dilated. It is necessary to use a blunt needle. Cistern punctures are rather easy to do, and the patients very rarely ever complain of headache; much less than in the lumbar punctures. Notwithstanding that many thousands have been done without any difficulty, I still feel some trepidation and anxiety when doing that puncture.

With regard to serum, I should say that the patients do not like it. Our patients object to it a great deal more than they do to ventricular or lumbar injections.

One final point: I am very much interested in any one who has an optimistic remark for the patients. More and more the people who try the hardest are the most optimistic. Certainly we still believe some of the patients are helped.

Dr. Percival Bailey: I think Dandy simply does what we have seen Dr. Cushing do for many years, during a cerebellar operation. As a matter of fact, the posterior route is a very bad way to reach the ventricle because the ventricle is far away from that point, and one is aiming at a very small part of the ventricle. I think it makes little difference where one punctures the ventricle. We never take measurements because if one puts a needle almost anywhere on the upper surface of the head, one will hit the ventricle provided the needle is introduced perpendicular to the surface. If the ventricle is dilated, one cannot miss it. I have never seen any trouble from puncturing the choroid plexus. Dr. Davis of Chicago, who is present this evening, can doubtless tell us about the posterior horn.

Dr. Loyal E. Davis, Chicago: Retzius, many years ago, called attention to the fact that in about 33 per cent of all human brains the posterior horn of the lateral ventricle is rudimentary and resembles somewhat that of the dog. I have recently confirmed this observation by filling the ventricles of brains properly hardened with mercury and then taking x-ray pictures of the specimens. This will explain the difficulty experienced at times in performing a ventricular puncture through the occipital horn particularly by those not well acquainted with the normal anatomy of the ventricles. I quite agree that ventricular puncture at almost any point in the cerebral cortex overlying the ventricles should prove quite simple at all times and it makes little difference as to the choice of the site of puncture.

Dr. F. J. Farnell: Merely as a matter of record relative to this ventricular procedure and the results derived therefrom; four or five years ago I treated, at the Providence City Hospital, six cases of paresis by the ventricular route. All were treated over a period of several months. I injected the serum into the anterior horn in all

cases. Two of these cases have since died, one is now in the State Hospital for the Insane, and three are attending to their duties and are at home. I had the pleasure of seeing two of the patients a few months ago, and both are in fairly good mental and physical condition. Those two patients have not received any form of antispecific treatment during the past two years.

Dr. A. H. Ruggles: The anatomical discussion I do not feel qualified to enter into. If we were to take the textbook plates and some work Dr. Wyatt has done on frozen sections, it would seem that the posterior horn was easier to enter than the anterior horn, and the distance is somewhat less; we have felt, therefore, that the posterior route offered fully as simple and certainly no more dangerous a procedure than the anterior route. I agree with Dr. Bailey that it is a simple procedure no matter which way one may enter.

Regarding Dr. Farnell's report as to the treatment: We have been following this a good while, and have spent much time and medicine on treatment, and yet we feel inclined to go on with it. I can't say we have cured any of our patients. Some of our remarkable improvements went to work two or three years ago; two are now dead; but every year it seems to me we send back to their jobs more and more patients. It seems to us that if we keep more of the patients to whom we give the intensive treatment on their jobs somewhat longer, it is worth while. To give a practical illustration: A patient who had an active period of paresis, following treatment was able to settle up his business, which amounted to three hundred thousand dollars. He sold out and invested his money himself, and though now ill again he was able to protect himself and his family.

It has been said that the good results of the treatment of general paresis now being reported are simply because the physicians and nurses are more interested in them, see them more often, and examine them more often. If that is the result, we had better keep on treating them. I believe with Dr. Farnell that some of the cases we have treated have done better than any we saw before treatment. We have one man at the present time who has been employed since early last spring on very difficult work as a machinist. When we saw him two months ago he was doing good work and getting on very well. Another man, a stonemaker, had his last treatment two years ago, and is still earning his living as a stonemaker.

SCHWEIZERKÄSE GEHIRN

DR. HUGO MELLA

The brain I am demonstrating this evening is one that perhaps may recall the war experiences of some of the members.

It is from a male patient, age forty-nine, who was first seen in the Nerve Department of the Massachusetts General Hospital in March, 1922. There, a diagnosis of cord tumor was made. Operation was performed at the Anna Jacques Hospital, Newburyport, by Dr. W. J. Mixter, who found a tumor in the center of the cauda equina. It was removed without any apparent destruction of the nerve roots.

Recovery was excellent except for a slight motor disturbance in one leg. The patient got on very well until August, 1923. On August 10 he quite suddenly became unconscious and was taken to the Newburyport hospital. At that time he showed an indefinite paresis of the extremities with rigidity of the neck. Two days before death he developed a severe phlegmon of the face extending from the hair line down over the forehead to the upper lip. Dr. Ayer, who will discuss the paper, punctured his cistern. On the 17th of August he died.

Necropsy limited to the head and cord was performed seven hours post mortem.

The upper half of the face was extremely edematous, of such degree that the eyes could not be opened. There was no crepitation in this area. The scalp was not adherent to the calvarium, nor was the dura adherent to the calvarium. On opening the cranium the dura over the left hemisphere was found to be very tense, whereas that over the right hemisphere appeared to be under normal pressure. On reflecting the dura over the right hemisphere the gyri appeared normal, whereas on the left, the gyri were flattened to such an extent that the sulci appeared as mere lines. The pia was hyperemic over both hemispheres. On tipping back the brain to sever the optic nerves, the left occipital lobe ruptured and a clot of dark blood about three inches in diameter was extruded; accompanying it were about two ounces of a thick, greenish, purulent substance. The brain was then removed and placed in formalin. The cord was removed with difficulty owing to the scar of the operation but was found intact.

After fixing the brain for six weeks, transverse sections were made when the striking condition, of which I show you the specimen and photographs, was found. The brain has the appearance of Swiss cheese, being "full of holes." These were also observed in the cerebellum and pons but not in the cord. Sections of the brain tissue show clumps of what are evidently gas forming bacilli. In the cortex were found ameboid glia cells, marked proliferation of glia nuclei, neurophagia, and clumps of fat in the neurones. No perivascular exudate was found in the sections studied. In the cord the only changes observed were occasional clumps of fat in the anterior horn cells.

I have also several photographs of a similar brain which Dr. E. W. Taylor had in his collection. In this brain the gas forming bacilli were also found.

Discussion: Dr. Percival Bailey: When I was an intern on the neurological service at the Cook County Hospital, Chicago, there came into the hospital a house servant who had had an abortion performed secretly a few days before. Her employer took her to a physician to be examined, and it was found what had been done, and also the condition from which she was suffering at the time—a very violent uterine infection. She was semicomatose on admission. While she was on the service she developed definite crepitation in the subcutaneous tissue practically all over the body. She died and the necropsy was reported thoroughly by Dr. Hassin in the *Archives of*

Neurology and Psychiatry, with an extensive discussion of the condition. At necropsy the brain was definitely crepitant. I don't remember how long *post mortem* the necropsy was performed. There was much discussion as to whether the change was *ante-* or *post-mortem*. Dr. Hassin felt strongly the changes were *ante-mortem* and he so published it. I think there can be no question that the changes were *ante-mortem*, though it was a considerable time *post-mortem* before the brain was taken out; and also that the presence of these lesions in the nervous system were not due to fixation in the formalin. In this case there were found the same organisms as Dr. Mella found in his case.

Dr. Raeder: In the Waverley Series we had a case which we called the cheese brain, but it was apparently due to a different cause. The case, about nine years old, was carefully studied by Dr. W. T. Councilman at the Harvard Medical School. It is reported in the second volume of the Waverley Research Series. There was a perivascular growth of a loose cellular tissue with perivascular edema and some mononuclear infiltration, and there was connected with it considerable hydrocephalus. The Wassermann was not taken in that case, but most of the histologic evidence pointed to syphilis as the cause. While this case gave the same picture, I noted that in Dr. Mella's specimen the holes are fairly general in the white matter and escaped the cortex, and that is the same picture that we had in our case; but while the condition in our patient was a chronic one, in this case, I take it, it was acute, although the gross picture is very similar.

Dr. F. H. Packard: I have seen a number of these brains and I think you will find that in the large state hospitals they are not infrequently seen. Some years ago we had such a case at McLean Hospital and the case was reported by Dr. Emma Mooers. The patient was a general paralytic well advanced. He rather suddenly became seriously ill; there was great distention of the abdomen; he became stupid and died. At the necropsy, which was done within twenty-four hours, the chest and abdomen were crepitant, in fact, more or less so everywhere except on the scalp. The pictures show the holes which were seen throughout the brain on section. There were similar holes in the liver and spleen and microscopically they could be seen in the kidneys and heart. I think that these changes are generally regarded as *post-mortem*; one reason for this being that the cavities have no membranous lining. Such cases were first described back in 1870 and there was for some time considerable controversy as to whether or not the changes were *ante-* or *post-mortem*.

Dr. Percival Bailey: In the case of bacillus infection that I saw, there was a definite pathological membrane surrounding some of these areas, and the areas were full of gas bacilli.

Dr. F. J. Farnell: I would like to ask Dr. Mella if there was any pathology in the brain aside from the abscess; and also whether the fat that was seen about the area of necrosis might not be the remains of a large group or area of fat cells destroyed? A recent report from England in relation to fat definitely states that formaldehyde acts on

some tissues and with some organisms by removing the fat and leaving a definite loss or changed area such as is seen in these pictures.

Dr. D. J. MacPherson: May I ask whether in the investigation of the first brain it was noticed whether these holes were around the vessels?

Dr. J. B. Ayer: This patient presented a clinical picture of meningitis. I was asked to see him with view to treatment via the cisternal route because lumbar puncture had failed to yield fluid. A blood-tinged fluid was obtained from the cisterna magna, which showed an increased number of cells, though not an excessive number, but no organisms either on direct film examination or on culture. Anti-meningococcic serum was given by this route, and repeated the next day, so strongly did we feel that a diagnosis of meningitis was correct. I observed no crepitation of the tissues on either day. In view of the possible infection with gas bacillus by the two cisternal injections it should be stated that all of the instruments used were boiled immediately previous to puncture. The serum administered on the first occasion was from the Mulford laboratories, on the second from the Massachusetts State Board.

Dr. Mella: The fat I spoke of was in the neurones of the anterior horns of the cord. As to the brain Dr. Raeder spoke of, I may be mistaken but I thought that was grouped under the degeneration we see in the idiot now and then. It may be related to the condition Dr. Courtney spoke of. It is probably due to a vascular lesion. I cannot find that these cavities are related to blood vessels; it is apparently mostly a diffuse involvement of the brain by these organisms. It did not involve the cortex to any extent but was not confined entirely to the white matter.

NEW YORK NEUROLOGICAL SOCIETY

THE FOUR HUNDRED AND SEVENTH REGULAR MEETING OF THE
NEW YORK NEUROLOGICAL SOCIETY, NOVEMBER 13, 1923. JOINT
MEETING WITH THE NEUROLOGICAL SECTION OF THE ACADEMY
OF MEDICINE

DR. E. G. ZABRISKIE and DR. HENRY A. RILEY, presiding

SYMPOSIUM ON EPIDEMIC ENCEPHALITIS

- I. Cases from the New York Neurological Institute.
- II. Cases from the Vanderbilt Clinic.
- III. Cases from the Neurological Service of the Montefiore Hospital.

I. Cases presented by Dr. E. G. Zabriskie, from the Neurological Institute:

Case 1. Female, middle age. Patient had typical onset, high temperature, delirium and then went into phase of acute bulbar

syndrome; was unconscious for six weeks. The temperature was 102.4°. There was difficulty in swallowing with a complete bilateral facial paralysis, and respiratory disturbance; she suffered very little pain, but was left with an extensive deltoid paralysis of both arms. The disease picked out different groups of muscles; on the right is seen a better extension of the forearm than of the fingers; on the left there is lack of extension of the forearm and good extension of the fingers. The cranial nerves have completely recovered, and she is making fairly regular progress between the upper dorsal levels.

Case 2. This case presents several unusual features. The young woman was taken ill four years ago when she was single. There was a typical course. She was ill for six or eight months with lethargy, diplopia, temperature. She had an inconstant fine tremor from which she apparently recovered so completely that she got married. Shortly after marriage she became pregnant and nine months later had a child. Six months after that she began to show the condition from which she is now suffering,—a tremor of the left arm and a peculiar facial expression, marked by a constant depression of the lower jaw which looks like a yawning reflex. She also has an enormous gain in weight, about 60 pounds, and this is still increasing.

Discussion: Major Jarvis (by invitation) asked: How frequent are the yawning movements? Does she sleep profoundly, or does this motion keep up at night?

Dr. Mailhouse asked: Has the patient been influenced by any form of treatment?

Dr. E. G. Zabriskie (closing) said: She sleeps fairly well. I have rarely seen the full yawning reflex. I have seen involvement of the platysma, of the sternomastoid, and of the diaphragm, but most frequently there is simple dropping of the jaw with partial involvement of the sternomastoid. In this case there is partial closure of the right eye. It is difficult for me to determine how much is voluntary movement to overcome the disability. She apparently made a complete recovery originally. No form of treatment has been of use.

Dr. J. W. Stephenson presented from the Neurological Institute a man, aged thirty-five years, watchmaker by occupation, whose remarkable improvement was considered to be the result of a definite line of therapy. This man entered the hospital the 18th of March, 1922, with violent myoclonus of the right upper extremity, the right abdominal muscle and the left lower extremity. So severe was the myoclonus that it was necessary to place boards on the side of the bed to prevent him from throwing himself out of bed. He was seen independently by several observers, all of whom agreed on the diagnosis, namely, epidemic encephalitis.

On entering the hospital the patient ran a temperature ranging from 100° to 102°. On March 23, strychnine nitrate gr. 1/30 with

pituitary liquid (Armour & Co.) minims $7\frac{1}{2}$ was initiated. His temperature at once began to drop and in five days became normal. Within forty-eight hours after beginning this therapy there was a marked subsidence of the myoclonus which completely disappeared in four days. It was then observed that the patient was developing a suspicious Parkinsonian attitude. The patient was kept upon the above therapy and discharged from the hospital April 24, 1922. He was kept under bi-weekly observation for six weeks and there was a gradual subsidence of the Parkinsonian attitude and gait. He was then allowed to return to his work—that of a watchmaker—and has been constantly at work ever since, in all seventeen months. The presentation of the case showed no abnormalities except a possible left facial paralysis of peripheral type.

Another case reported but not demonstrated was that of a young man, tailor by trade, aged thirty, who entered the hospital September 8, 1921. He was a very ill man, with multiple cranial nerve involvement, and had developed a persistent vomiting which caused him to be put on the "most critical" list. For the first four days in the hospital his temperature varied between 98° and 99.6° , then suddenly dropped to 97° and for eight days varied between 96° and 97° . For the first fifteen days he was given only strychnine nitrate, and on the fifteenth day pituitary liquid (Armour & Co.) and at once his temperature began to rise and in eleven days his temperature by steady progression reached 98° , and when he was discharged from the hospital it varied between 98° and 98.6° . Whereas the vomiting had been a critical symptom prior to the administering of the pituitary liquid he vomited only once after its initiation, that being the second day. From then there was a slow but steady subsidence of all symptoms and on the 6th of December, 1921, the patient was discharged, having practically no cranial nerve involvement, but a suggestion of the residual right hemiplegia. Within a month's time he was back at work, and has been constantly at work ever since, now twenty months. He now shows no objective findings and has no complaints.

It was stated that this particular line of treatment was carried out in about ten cases. The cases in which it was believed favorable results followed were the acutely ill, the spinal type with marked myoclonia, and the cranial nerve with diplopia type. In the acutely ill and myoclonic types improvement invariably began within forty-eight to seventy-two hours after the beginning of treatment. In the cranial nerve with diplopia type, progress was slower. The cranial nerves showed improvement within a week to ten days and the diplopia disappeared within a month to six weeks. The frankly Parkinsonian cases were not influenced at all.

Whereas nothing miraculous was claimed for this particular therapy, yet the regularity of the time of appearance and the uniformly similar progress of the improvement was not considered a coincidence, but a consequence of said therapy, and inasmuch as it is harmless it was thought worth while to present these facts to the Society.

*II. Cases from the Vanderbilt Clinic:*CASE OF POST-ENCEPHALITIC PARALYSIS AGITANS
SYNDROME, WITH UNUSUAL FEATURES

DR. RUBIN A. GERBER (by invitation)

The following case is presented because of the interesting affection of the musculature of the head, a movement approaching what has been analyzed as characteristic of dystonia, varying from the usual hyperflexion of the body musculature seen in paralysis agitans by presenting a hyperextension of the muscle of the head segment.

F. S., male, twenty, presented, on October 20, 1923, shaking of the left hand and stiffness of the neck. He said that he had had "influenza-neuritis" in January, 1920, for three months, the condition being brought on by exposure shoveling snow; and it was characterized by delirium, sharp shooting pains down the left arm, and insomnia for three weeks. This was followed by a period of somnolence of several weeks duration. For two years, until February, 1923, he considered himself in good health with no sequelæ of his disease, except a very slight tremor of the left hand. He worked and had no other complaints until one day in February, 1923, he was struck by a taxicab. He was dazed, turned white and trembled all over. Apparently he was unhurt, though shocked, and he was sent home in a taxicab. That same day he began to notice a violent shaking of the left arm, at times so great as to shake his whole body (the condition he shows at present). Gradually, since then, his head began to be drawn backwards against his will and with difficulty he is forced frequently to flex his head forward. Slowly, involuntarily, and gradually the head extends backwards again, until there is a disturbance in circulation, the face becoming suffused, the blood vessels of the neck engorged and the muscles strained to the utmost. The mouth remains open and there is marked tremor of the tongue which makes a flapping sound. Mucus and salivary secretion are increased, causing slight choking and drooling. He has observed occasionally a slight trembling of the right arm and right foot. Since attending the clinic he has occasionally had "fainting spells," not losing consciousness, but falling to the ground. He becomes blue in the face, froths at the mouth and trembles all over. These attacks last for about five to ten minutes with no incontinence but followed by marked malaise. Lues denied; habits regular; no noteworthy family history. On examination he shows marked abnormal attitudes; a marked irregular tremor of the left hand when at rest, which, by its violence, causes the whole arm and left side of the body to shake; a flexed attitude of his skeletal musculature, but his head is held backwards stiffly. In a supine position his feet are extended and somewhat internally rotated. He has a propulsive gait with marked latero-pulsion. When standing, he holds his feet together only for a few seconds and then is involuntarily forced to take a step or two backwards to balance himself. There is a lack of the associated movements of the left arm and the head when waking, and a slighter

repression of the associated movements of the right arm. He sits down in one movement—all in a lump. Though there is no definite Romberg, he sways considerably and is retropulsed. When sitting for a few minutes, he gradually falls to the left and on one or two occasions has fallen from the chair. There is a marked tremor of the left hand when placing finger to nose and also when placing finger to finger. He has slight adiadochokinesis of the left hand and some slight dysmetria. He writes with the typical P.A. incoordination. Speech is dull, weak, and monotonous. Dysarthria is increased by the tongue tremor and excessive mucus production and salivation. There is tremor of the lids and of the lips. The head assumes a hyperflexed attitude by a gradual slow dystonic-like movement of the head. There are no pathological reflexes. Muscle strength and muscle status are normal. There are no abnormal associated movements. Nerve status is normal. Sensory examination is negative. The left pupil is slightly larger than the right and both the right and left show a slight diminution of the direct light reflex. There are some slight nystagmoid jerks when the patient is lying down and looks to the left. Typical paralysis agitans facies with a flattening of both sides of the face is present and the smooth glossy skin and increased salivation. Mental status is apparently normal as regards intelligence, memory and attention, but there is marked susceptibility to emotional stimuli. Viscera normal. Pulse 80. Inanition beginning. Laboratory tests were negative as regards urinalysis, blood Wassermann, and spinal puncture.

The several points of particular interest in this case are: (1) The cerebellar phase, with the presence of both intention and volitional tremor, the nystagmus, festination and disturbance of gait. (2) The presence in a case of encephalitis with paralysis agitans syndrome, of a dystonic-like movement limited to the head segment. (3) The relation of trauma, with the incidental emotional upheaval as bearing on the production of the acute phase of the disease.

We might analyze the pathology as a fragmentation of various circuits, showing hyperextension of the muscles in one segment and flexion in the others. The cerebellum, striate system, static and kinetic mechanisms and perhaps the cerebrum are involved. The latter through pronounced irritation or the lack of proper inhibition may be the cause of his fits.

Discussion: Dr. Abrahamson asked: How long after the accident did the patient note the increase of symptoms?

Dr. Smith Ely Jelliffe asked: Is there any glycosuria, levulosuria, galactosuria, or any blood chemistry change? Have you noticed hyperthermia or changes of the sweating mechanisms?

Dr. E. D. Friedman said: We have had a similar case at Bellevue. There was hyperextension of the head and sialorrhea. Such cases indicate that there must be segmental innervation in the basal ganglia. All of us who have seen encephalitis will recall cases with focalized rigidity and tremor in one limb or in a particular muscle group. These cases can only be explained by the assumption of segmental innervation mechanisms.

Dr. Gerber (closing) said: We made only routine examinations of the blood and urine, and these showed nothing abnormal. The only secretory changes are the glossy appearance of the face, the increased salivation and increased mucus production. No hyperthermia has been observed. The increase in symptoms was noticed immediately after the accident.

CASE PRESENTATIONS FROM THE NEUROLOGICAL SERVICE OF MONTEFIORE HOSPITAL

BY DR. S. BROCK

Case 1. Unusual paralysis agitans following acute epidemic encephalitis, showing a diurnal variation in motor activities. A. P., female, eleven years old, was taken ill February, 1920; abnormal movements presented themselves which ceased only during sleep. On the third day a fever developed which gradually rose to 104° and she saw double. The next day drowsiness appeared, which increased to lethargy after about five or six days. She was incontinent. The lethargy lasted about six weeks and it was noted that a Parkinsonian gait and attitude had developed together with a well-marked tremor in the head and upper extremities; speech became low-pitched and monotonous, until, finally, about November, 1921, she became almost entirely inarticulate and swallowing became very difficult. *Physical examination* reveals a marked Parkinsonian rigidity in which her head is fixed in a somewhat retracted position and turned to the left; the upper extremities are somewhat adducted at the shoulders, flexed at the elbows and wrists. The lower extremities are adducted at the hips, slightly flexed at the knees; the right foot is in marked equinovarus, the left marked equinus. Everywhere there is the "cogwheel" spasticity. She lies in the recumbent posture and seems incapable of the slightest voluntary movement. *Cranial nerves:* The eyes are turned upward and to the left, the pupils are round, equal, react rather sluggishly to light and accommodation. Ocular movements are fairly well performed. The fundi are negative. There is the fixed countenance of paralysis agitans with greasy skin. She is unable to protrude her tongue. There is drooling of the saliva. Swallowing is accomplished with considerable difficulty. In fact, there is a pseudobulbar palsy of striatal type. There is a constant coarse rhythmic tremor of upper and lower extremities which varies in amplitude from time to time. The deep reflexes are quite lively, the right more than the left. There is bilateral ankle clonus, left greater than right. There is no Babinski. There are no sensory changes. During the day the child lies like a piece of statuary, immobile except for the tremor. At night, usually after dark (9 to 11 p.m.) she arises from the bed, walks and is said even to have run about the ward. Speech also is said to have returned in part. These latter manifestations point to a remarkable mutation, which encephalitis has been known to produce in the sphere of sleep. (At Montefiore Hospital, another case of paralysis agitans, following

epidemic encephalitis, in a young boy of seventeen, showed at times a remarkable condition somewhat allied to the above. He would gradually "fold up" in his Parkinsonian attitude, falling from an upright position to a crouched, stooped one, and yet when given the command to get up and walk or run, he would do so in a most remarkable manner, which contrasted markedly with the severe paralysis agitans present.) The examination of blood, spinal fluid and urine is entirely negative.

Case 2. S. G., male, forty-two years old. Past history irrelevant. Present illness began in 1920 with headaches, double vision, marked insomnia, and fever. He "recovered" and returned to work. One year ago he noticed that he could not work as fast as formerly and that there was a certain stiffness and pain in the back. Six months ago he began to complain of a tremor in his hands and to a lesser extent, his feet. *Physical examination* reveals a typical Parkinsonian facies. The gait is normal, except for the loss of associated swing of the upper extremities which are held in the typical Parkinsonian posture. *Cranial nerves:* Slight blurring of left fundus. Slight left ptosis; wide palpebral fissures; prominent eyeballs; definite bilateral von Graefe. The movements of the eyes show "cog-wheel" phenomena. There is weakness of both external recti muscles, and the eyes converge scarcely at all. The pupils are equal, react fairly well to light and accommodation. The left side of the face is slightly parietic. The tongue protrudes slightly toward the right and is somewhat tremulous. The left palate seems to hang lower than the right.

There is a very curious smacking movement of the lips associated with which is a swallowing reflex. This movement occurs at the rate of about five to ten a minute. It ceases when the eyelids are closed and also upon command. The patient volunteers the statement that he performs this movement because of dryness of mouth and tongue. There is a marked rhythmic tremor of the hand. While the deep reflexes are generally increased there is no clonus or Babinski; the superficial reflexes are very lively. There are no sensory disturbances. The face shows a greasy skin. The mouth does not show an undue dryness.

Discussion: Dr. Stephenson asked: Has she had any form of treatment?

Dr. Brock said: She has had nonspecific protein therapy. She is to get typhoid vaccine.

Dr. Riley asked: Can you describe the circumstances under which she regains voluntary control?

Dr. Brock said: I have not witnessed this myself, but the internes have seen it frequently.

Dr. Abrahamson said: This case is one of the group I presented at the Boston Meeting of the American Neurological Society. The phenomena and causation of the phasic and permanent mutations in lethargic encephalitis were discussed and analyzed. The frequent nocturnal mutations are of special interest; this conversion of the activities of the day into those of the night and *vice versa* have

been noted by many observers. Mutations for a time phasic may eventually resolve themselves into permanent changes. Syndromes with all appearances of permanence, may remit and remain absent for an indefinite time; or it may appear in a different form on the next occasion. In the phasic alternations, we must predicate the intactness of the upper and lower motor neurons, of the motor apparatus; paralyses in the usually employed meaning, cannot exist there; otherwise such excellent movement would never be possible. The will to move is affected; the central organ of tone suffers. When all things are favorable, action is possible; when the mood or other circumstances are adverse, such movements are missing. It is a serious mistake to regard these patients as hysteric, or worse, as malingerers.

Dr. Henry A. Riley asked: Is there any synchronism in the movements between the right upper extremity and the left lower extremity? It appears as if there were. This would be interesting in relation to the crossed motor association as seen in quadrupeds, between the right fore foot and left hind foot, and *vice versa*.

Dr. J. H. Leiner said: I would like to know if this child speaks at night. In one case seen, of a little girl, the child kept up continuous talking from 8 P.M. to 5 A.M. It was practically monosyllabic repetition: "Mummy, are you asleep? Daddy, are you asleep? I'm going to sleep," over and over again. The parents thought it was hysterical and tried to break her of it by punishing her. They could not stop her. This child is a typical post-encephalitic Parkinsonian case. She has gained 50 pounds in weight.

Dr. Brock said: I am told this girl articulates at night, but in the day time it is impossible for her to talk.

Dr. Smith Ely Jelliffe said: It is of considerable interest to follow Dr. Abrahamson's suggestions relative to the innumerable fascinating problems which modern insight into the anatomy of the nervous system has brought into prominence through the encephalitis symptom-complex. Some here may recall Vogt's interesting discussion of the striatum syndromes and particular attention may be directed to his thesis in the Heidelberg Akad. der Wiss., 1919, in which he emphasized the value of an understanding of the striatum regions as a possible structural substrate of many emotional reactions. Hysteria was particularly mentioned. Kapper's¹ recent study of the phylogeny of the striatum in birds and the relations of these to that region in man has advanced the question a step further. He has made it more clearly appreciable that some day we may be able to talk of the "anatomy of the unconscious," *i.e.*, the fiber connections that hitch up the older affective processes relative to the vegetative life of the "organism as a whole." In this study he more closely points out the fact that the "paleostriatum" in some of its nuclear and fiber arrangements is to be considered as an outgrowth, not of the diencephalon, but the telencephalon or cortex. Here may be traced the anatomical ways through which the cortex, through so-called psychical function, is intimately bound up with the vegetative or affective craving portions of the body. So much for ana-

tomical considerations. As for the psychological situation, you are all familiar with Janet's older generalization concerning the "lowering of the tension of the psychical level." This is what happens with this type of case, speaking in a general manner. The war experience showed this, and a great number of experiments bear on the issue. These may result, either from direct injury, inflammations, emotional shock, toxemias, etc., the cortical *balancing* factors are removed and the primary vegetative factors come to the surface. Then, during the night time, or under any influence that narrows the issues to primary stimulus, the cortical regulating factor upon the disordered motor mechanism is partially reestablished. Thus these patients are apparently well at night, or they can dance, or sing in response to the stimulus of a graphophone, a piano, a funny story, or anything that releases a simple emotional response. The paleostriatal motor coordination pathways are for the time surcharged with the affective tension, get to the cortex via the pathways indicated, the which are only partially destroyed structurally, and the peculiar motor manifestations, or mutations, as Dr. Abrahamson terms them, are modified, or disappear. I would call your attention here to Verger et Hesnard's,² Pette's,³ Salmon's,⁴ and Lewy's⁵ studies in this connection, where these bradykinetic phenomena are exhaustively considered, not to mention many others already cited in the last edition of Jelliffe and White, *Diseases of the Nervous System*, 1923.

Dr. Brock said: This patient is able to stop the movements of the mouth when given the command.

Dr. Abrahamson said: That means really that it is an early stage. Later he will not be able to respond to command. He will lose control.

Dr. Craig said: It would be interesting to see if he can still move the mouth when keeping his eyes closed.

Dr. Smith Ely Jelliffe said: I would like to emphasize what Dr. Abrahamson has said. His point is extremely interesting. This whole series of phenomena reminds us that we have been too much inclined to regard the human being as a machine working by itself of itself, and have forgotten how far it is related to the environmental stimuli. We must regard the individual as a transforming mechanism. The environment is always playing upon the individual. In the average physiological state we are able to handle the stimuli. The phenomena not only involve the associated movements, but the cutting out of stimuli that are always falling upon the nervous system force a new state of adjustment. A diaschisis results (v. Monakow). In these patients stimuli are cut out because the organism is not able to handle them. If you can remove the stimuli that overcome them, the phenomena disappear. This may be a mechanism of defense, as the turning off of the stimuli may help the organism to regain its equilibrium.

Dr. Joshua H. Leiner said: The question of therapy concerning epidemic encephalitis arose this evening. During the early days of the epidemic in this country, a belief arose that thecal puncture

improved the patient. This view was short-lived. Very little, if any, therapy was resorted to at this time.

Later, reports began to infiltrate from the other side (chiefly from France) that nonspecific therapy was in use. Marinesco used serum intraspinally, Döllkin used milk. Brill, in this country, was using serum intraspinally. A well-known bacteriologist from a western institution has been using a horse serum which he believes is specific. Its use in two of my cases was followed by severe anaphylaxis, one case showing alarming crises, vagal in type. She died a week later. Judging from the favorable reports that emanate from this investigator, it is my conviction that his good results are in the main due to nonspecific proteins within the serum.

Three years ago I used turpentine for the production of a fixed abscess in a woman, who was in a lethargic state for five weeks in St. Mark's Hospital, and three weeks on our neurological service in Lebanon Hospital. She began to lose very rapidly. Her vasomotor system was failing, and her blood pressure dropped to an alarming level. Her condition being very precarious, as a last resort we produced a fixed abscess. Her change was most dramatic. In three days her eyes were wide open, and in nine days she was in a wheel chair. She was then sent to the Montefiore Hospital to convalesce, and is now well. The production of antibodies due to a fixed abscess, which is an old remedy, improved this condition.

A case of epidemic encephalitis in a child showing sequelæ of respiratory and conduct disorders (J. A. M. A., Oct. 13, 1923) got better spontaneously, following an attack of lobar pneumonia. The production of antibodies on the basis of nonspecific reaction improved the child.

As a result of these observations I began to treat my patients on the basis of nonspecific therapy, using a nonspecific protein in the form of sodium nucleinate (Merck). With a preparation of this type of protein we have the advantage of graduating the dose, and thereby control the phase of anaphylaxis. Then again, its pharmacological and physiological properties are well known, even preceding Netter's use for the paralytic diseases, *i.e.*, the production of leucocytosis and phagocytosis.

I have used this now in over 25 cases with very favorable results. Nineteen of these cases were in the acute-subacute stage of the disease. This nonselected group were all severe cases entering the wards of Lebanon Hospital. One fatality took place, and this was in a case in which treatment was first started three weeks after its onset. In the acute stage we begin with a half grain subcutaneously, watching the reaction, *i.e.*, temperature, pulse, total and differential white count. In the subacute, chronic variety, where a fixed pathology is already present, one can hardly expect much, except to attempt to stay the progress of the disease.

In this disease there is no frank reaction on the part of the organism, as is evidenced by the lack of leucocytosis, with its oft accompanying phagocytosis. Spiller and Ayer have made note of this fact, *i.e.*, the lack of leucocytosis. A reaction of the host to an

invading organism, creating the production of antibodies, is an all important item in an infection. Our specifics, such as salvarsan, and quinine, have the property of producing leucocytosis. In the early days when intravenous salvarsan was combined with subcutaneous injections, the Wassermann reaction disappeared much earlier, this being due to the greater leucocytosis. The use of quinine in puerperal infections is given for the same reason. Wagner-Jauregg's treatment of general paresis is nonspecific therapy.

I am frank to say that I do not know what my results will be in years from now. I know of two severe cases that I have had under observation for nearly two years. One a hard-working U. S. mail carrier, who is out in all weathers, and performing his usual duties. The other one was a patient of the late Dr. Leszynsky, whose condition was so desperate at the time that he felt very little harm could be done if this new therapy was given. He had a very severe reaction after the first dose, but recovered, and is now working as a tailor. In regard to the good results following the use of pituitrin, as shown by Dr. Stephenson, I believe the effect is due to a protein. When strychnine nitrate was given alone, there was no improvement, but given with increasing doses of pituitrin, there was improvement. I believe this is a nonspecific result.

I think we ought to try and do something for these patients, instead of leaving them alone, until a specific remedy arises. We should use our armamentarium as physicians, and do what good we can to these unfortunates. Surely no harm can come from the above procedure.

Dr. Friedman said: I should like to dwell upon the lessons of the encephalitis epidemic and to stress one or two physiological considerations. In the first place, the encephalitis epidemic has taught us the important rôle which the basal ganglia play in the genesis of abnormal involuntary movements. These great structures are no longer the terra incognita of the brain. Prior to the encephalitis epidemic, many of the bizarre movements which could not be explained were labeled hysteria. We now know that most of them have an organic basis. Observation and study of the post-encephalitic sequelæ have confirmed the work begun by the Vogts and Foerster in Germany, and carried on in this country by Hunt, on the extra-pyramidal syndrome. We now know that most of the Parkinsonian syndromes are due to lesions in the globus pallidus, and that the putamen-caudate or striatum proper is the site of the pathological process in cases presenting choreiform and athetoid movements. Probably the dystonias also belong in this group. The substantia nigra has come prominently to the fore in recent pathological studies.

The encephalitis epidemic has taught us further the importance of the midbrain as the great vegetative center. The lethargy and sleep disturbances noted in encephalitis both during the course of the disease and as sequelæ, confirm the theory of Troemer, who postulated a sleep center in the midbrain. It is disturbance in function of this center which leads to stupor in polyencephalitis superior (Wernicke). The profuse sweating, the greasy face, due to

increased secretion of sebum, the disturbances in metabolism, often leading to marked increase in weight, the sialorrhea, the frequency of bladder symptoms, particularly in the acute stage of the disease, the respiratory disturbances noted often during the later period and the presence of tachycardia in many of the cases in spite of the relatively low temperature—all emphasize the significance of the midbrain and hypothalamic region as the center of our vegetative life.

Dr. Abrahamson said: The cases presented bring up two very important problems: one, the relationship between accident or trauma and encephalitis; there is no doubt in my mind that accidents can later accentuate or initiate recurrences or exacerbations of lethargic encephalitis; it acts as an activator or accelerator of the morbid process; its effects are physical or psychical, especially emotional. The second point of interest relates to marriage and lethargic encephalitis. This subject must be met squarely; considerable unhappiness has resulted from the marriage of patients who have had a previous lethargic encephalitis, and who, for the time being, present little or no evidences of the disease. No one is in a position to predict when or in what fashion the disease may reappear as a chronic manifestation, neither can we tell any patient that a recrudescence is out of question. The specific action of the virus on the basal ganglia and the frequency of disturbances of the sexual sphere, along with the mental manifestations, all must make us very cautious in recommending marriage. The clinical resemblances between lethargic encephalitis and epidemic poliomyelitis must be borne in mind; the present rather mild, though fairly extensive epidemic of poliomyelitis, has presented many cases where a clinical differential diagnosis was well nigh impossible; the determining factor being that one was prevalent and the other was not. As regards therapy: in the acute and subacute stages 80 to 90 per cent recover from the acute illness apparently independent of therapy. Once the chronic manifestations, such as Parkinsonian or dystonia syndromes exist, little can be hoped from nonspecific or any form of treatment. Efforts should be directed with nonspecific therapy to prevent, if possible, the occurrence of chronic manifestations which therapy has been under way for the past three years.

Major Jarvis (by invitation) asked: What is the mortality over a number of years? I was reading a report of deaths last year. I do not see any deaths listed from encephalitis.

Dr. Grossman said: That is a difficult question to answer for any given year. Following the mortality over a number of years in the various epidemics, it is found greater in some than others. In a group of 145 cases observed at the Mount Sinai Hospital, over a three-year period, it is 20 per cent. I think that is actually too high because the cases were of a severe type. The average mortality would be nearer 10 than 20 if another group of cases were studied.

Dr. Stephenson said: In hospital statistics the mortality among adults is 10 per cent and among children 15 per cent.

Dr. Smith Ely Jelliffe said: It is necessary to go beyond the

narrower nosological considerations raised in a slightly overacademic matter by Dr. Abrahamson. He has spoken of the needs of closer clinical differentiation between what we have been pleased to call *encephalitis*, and what we have been pleased to call *poliomyelitis*. These may not, after all, be separable. If we go into the history of epidemics we might raise a question as to what we have been pleased to call *influenza*. From the fourteenth century on we have historical documents showing the presence of all these kinds of clinical medleys associated with influenza. By what differentiation are we entitled to say that these psychotic, neurological, psychological symptoms are due to influenzal poison, to encephalitis poison, or to poliomyelitis poison? These may be shifting types of reactions in the nervous system to a mixed virus which may be expected according to the degree of stress in which the individuals are involved. Here environmental factors of an important nature may have to be included. This is not merely my own hypothesis. Thus: Lépine of Lyons, at the Paris Congress of 1921, and others have raised the same issue, and have emphasized the possibility that the more intense types of reaction involving vegetative levels of life and which gave rise to what we are now calling encephalitis lethargica, might be better understood when the enormous economic stress throughout the entire world, the population being involved in much more acute conditions, have produced the type of disease we call encephalitis lethargica, *because* the vital affective pathway portions of the nervous system were under greater stress. The factor of predisposition of localization of a disease process, causing different clinical pictures, by reason of different environmental stresses became germane to the discussion. I have not infrequently called attention to this in the various syphilitic processes. In former years great discussions arose as to the differentiation of different diseases of the nervous system, which were all shown to follow one virus, syphilis. But why the process should be a paresis, a gastric crisis, a myelitis, that had to be relegated to other more obscure factors in the discussion of which the definite stresses of the affected organs needed to be incorporated. I have frequently mentioned the possibility of a rectal tabetic crisis being a resultant first of the syphilitic virus, but secondly located where it is because of a psychoanalytically understood perverse anal eroticism.

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CURRENT LITERATURE

I. VEGETATIVE NEUROLOGY.

2. ENDOCRINOPATHIES

Hastings, A. B., and Murray, C. D. EFFECT OF PARATHYROIDECTOMY ON BLOOD CHEMISTRY. [Jour. of Biological Chem., March 1921.]

The effects of parathyroidectomy on the calcium, sugar, combined carbon dioxide, and hydrogen-ion concentrations of the blood were studied by Hastings and Murray. The previously observed calcium deficiency in parathyroidectomized dogs was verified, but no support is found for theories based on a disturbed acid-base equilibrium. General theoretical considerations are outlined which may be of some value in explaining the relationship between tetany and the relative concentration of certain ions in the blood.

Editorial. THE BLOOD IN INFANTILE TETANY. [J. A. M. A.]

About 1 per cent of the weight of the protoplasm of the body is composed of inorganic elements. Of them Mathews¹ has expressively written: "They are not simply clinkers clogging the grates of the protoplasmic fires, but they are active in the production of the vital phenomena." In recent years, physiology has learned to regard the inorganic constituents of the body tissues not merely as structural units, such as one might assume the mineral components of the bone to be, but also as elements taking a more active part in relation to physiologic function. It has become apparent in numerous instances that any change in their relative proportions at once affects the activity of the cell; thus, by increase or by diminution in the proportion of sodium, calcium or potassium, skeletal muscle may be made to twitch rhythmically or to remain at rest; nerve impulses may be set up in motor nerves, or the irritability of the nerve raised or lowered. Hence has come the conception of a "physiologically balanced" solution representing the ideal circulating fluid of the organism—a fluid in which the relative proportions of the different ions is such that their antagonisms are neutralized and the proper balance for the maintenance of a normal irritability of muscle and nerve is secured.

The studies of Loeb and others have shown, for example, that calcium and magnesium are antagonists to sodium and potassium. Small amounts of calcium prevent the stimulating action of sodium in experiments on isolated muscles; hence Loeb once remarked that we are in-

1. Mathews, A. P.: *Physiological Chemistry*, New York, William Wood & Co., 1920, p. 14.

debted to the calcium concentration of our blood for the fact that our muscles do not constantly twitch. There are disorders of man in which the musculature either twitches or shows a hyperexcitability to electric stimulation. There has been much speculation as to whether and how such phenomena might be related to an "unbalanced" condition of the body fluids which perfuse the organs.

Conjecture can now be replaced by knowledge, because the inorganic composition of the blood is no longer as much of a mystery as it used to be. The physiologic chemist has devised methods of microanalysis by which even small quantities of blood can be made to tell the story of their chemical composition. From data obtained in this way, Kramer, Tisdall and Howland² have confirmed earlier evidence from the Department of Pediatrics at the Johns Hopkins University indicating that the concentration of the sodium, potassium and magnesium in the serum of patients with tetany is essentially normal. On the other hand, the concentration of the calcium is regularly lowered. Obviously, the Baltimore pediatricians conclude, the important factor in increasing the irritability of the neuromuscular mechanism in infantile tetany is the decrease in the calcium concentration. The stimulating effect of the sodium and potassium salts is unopposed by the inhibitory effect of calcium. A procedure for the treatment of infantile tetany thus immediately suggests itself.

Togawa, T. STUDIES IN THE METABOLIC CHANGES IN EXPERIMENTAL TETANY. [J. Lab. and Clin. M., 1920, V, 299. Med. Sc.]

In parathyroidectomized dogs, showing typical tetanic symptoms, a condition of acidosis is always observed. The antitryptic power and nonprotein nitrogen content of the blood are usually increased. There is no acidosis in thyroidectomized dogs showing no tetanic symptoms; a slight alkalosis is sometimes noticed. In these cases the antitryptic power and nonprotein nitrogen of the blood are unchanged from the normal.

Pincherle and Maggesi. SPASMOPHILIA AND CHANGES IN ENDOCRINE SYSTEM. [Riv. d. Clin. Ped., October 1920, XVIII, No. 10.]

This investigation was of seven children, six of whom had died from laryngospasm, and the seventh from tetany associated with pneumonia. All showed positive Chvostek phenomenon and frequent Trousseau. The Erb phenomenon was marked in four, and convulsions were frequent. The endocrine system was specially studied. The thymus was involved in all; the parathyroids in all but one showed more or less sclerosis, sometimes hemorrhages. The authors argue that the spasmophilic trend is sufficiently explained by their findings, to which they add a strong familial tendency to glandular defect transmission.

2. Kramer, Benjamin; Tisdall, F. F., and Howland, John: Observations on Infantile Tetany, *Am. J. Dis. Child.* XXII, 431 (Nov.) 1921.

Farner and Klinger. EXPERIMENTAL RESEARCH ON TETANY II.
[Mitt. a. d. Grenz. d. Med. u. Chir., 1920, XXXII, No. 4.]

Some animal experimentation researches, chiefly carried on with cats, by partial and total excisions of the thyroid and parathyroids. Acute or chronic tetany followed in the larger number of cases. In some, no symptoms were observed; in some others, the acute attack could be modified by a suitable calcium diet, and a state of tolerance to the condition established, interrupted by occasional acute onsets. These authors state that for the cat the presence of accessory parathyroids does not protect against lethal tetany. Rudimentary glands do not seem to entail chronic tetany conditions. The total removal of the thyroid may give rise to acute tetany; if a functioning portion is left, the removal of all four parathyroids may be carried out without symptoms. The use of calcium always stopped acute cases of undoubted tetany but the effects last but one or two days, hence the calcium must be continuously given. The experiments lead to the conclusion: They follow the Paton school in reference to the influence of guanidin and methyl guanidin, saying that calcium follows up the latter, but really offers no dynamic explanation of the phenomena.

Blühdorn. WHOOPING COUGH PLUS SPASMOPHILIA. [Zeitschrift für Kinderheilkunde, September 1920, XXVI, No. 5.]

It is imperative, the author states, to diagnose and treat spasmophilia in children with whooping cough. This element can be eliminated by withholding milk and giving cod liver oil and lime. Further meningeal complication from pertussis may occur. This is responsible for many of the spasmodic symptoms. This too calls for special treatment, reducing the pressure by lumbar puncture and giving a sedative. The benefit may be great when lumbar puncture alone or chloral alone has proved ineffectual. In a number of recent cases of serous meningitis in the course of pneumonia, including some with whooping cough, treatment of the spasmophilia materially mitigated the severity of the disease. A differential diagnosis between spasmophilia and whooping cough cannot always be made. This suggested to the author the withdrawal of milk and the use of cod liver oil. The withdrawal of calcium increases a spasmophilic tendency and thus light is thrown on the two possible components.

Priesl, A. A CONTRIBUTION TO THE KNOWLEDGE OF HYPOPHYSEAL DWARFISM. [Beitr. z. path. Anat. u. z. allg. Path., 1920, LXVII, 220.]

The author has had the opportunity of carrying out the post-mortem examination of a dwarf who had reached the remarkable age of ninety-one years. A bilateral lobular pneumonia had been the immediate cause of his death. Besides a recent cystitis, a moderate degree of arteriosclerosis and senile atrophy of most organs were observed. As to the

endocrine glands the following changes were noted: (1) Thyroid and suprarenal glands very small; (2) lateral parathyroid glands uncommonly large (true hyperplasia); (3) genital organs hypoplastic; (4) traces of an old and in part disappeared obesity; (5) important alterations of the pituitary body, the anterior lobe of which was extremely reduced in size, and its glandular parenchyma formed of only a few and incompletely differentiated cells; its posterior lobe was dystopic, viz., situated outside the *sella turcica*; the junction between the two lobes anomalous and incomplete. The author lays stress particularly on this fact, which probably had an injurious influence upon the nutritive conditions of the anterior lobe, and ultimately caused its atrophy, at approximately the age of sexual development. The primary cause of these alterations appear to have been the persistence of the cranio-pharyngeal canal and an anomalous ossification of the posterior half of the sphenoid. [Da Fano.]

Borchers, E. PARATHYROID TRANSPLANTATION. [Zentralbl. f. Chir., November 6, 1920.]

Owing to the fact that parathyroids are often confused with other bodies of similar appearance in dissections in the living animal such as accessory thyroid glands, lymph nodes, or fat lobules, this author maintains that many of the negative findings of parathyroid transplantation can be explained, and for his own work he has established a rule that no parathyroid transplantations be carried out without histologic control. The macroscopic appearance of the bodies encountered is an unsafe guide as to what they are.

Barrett, A. M. PSYCHOSIS ASSOCIATED WITH TETANY. [Am. Jour. of Insanity, April 1920.]

The author is convinced from an analysis of cases, that there is no specific tetany psychosis, but that the neuromuscular disturbances and the psychosis are both the result of a toxic process affecting the central nervous system. He reports two cases in which this disturbance seems to have had some relationship to disease of the pituitary.

Mittasch, G. ON HERMAPHRODITISM. [Beitr. z. path. Anat. u. z. allg. Path., 1920, LXVII, 142.]

v. Keussler, H. ON SOME CASES OF HERMAPHRODITISM. [Beitr. z. path. Anat. u. z. allg. Path., 1920, LXVII, 416.]

Poll, H. TUMORS OF THE INTERSTITIAL CELLS OF THE TESTIS IN BIRD HYBRIDS. [Beitr. z. path. Anat. u. z. allg. Path., 1920, LXVII, 40.]

Mittasch has had the opportunity of fully investigating a case of "true" male hermaphroditism. The generative glands had the form and situation of the ovary, but the structure of the testis. Most seminiferous tubules were lined by a stratified germinal epithelium, but no transformation into sperm-cells or fully-formed spermatozoa were

seen in any section of the serial preparations. Other tubules showed all degrees of atrophy and degeneration. The interstitial tissue was greatly increased and essentially formed of triangular or polygonal cells provided with large nuclei and rather dense cytoplasm often containing granules of brown pigment and droplets of fat. The accessory system of the male generative gland (rete testis, epididymis, vesicula seminalis, and prostate) was complete and sufficiently well developed. The vas deferens had a somewhat anomalous situation and, while opening in the vesicula seminalis as normally, ended blindly on the side of the epididymis. Of the female generative organs the Fallopian tubes, uterus, and vagina were present and rather well developed. The Fallopian tubes had no abdominal ostia; the uterus was formed as usually of a body and a cervix; this last continued with the rather short vagina which opened into the male urethra at the place where the prostatic utricle and verumontanum are normally situated. Fallopian tubes, uterus, and vagina were filled with coagulated blood, of which some existed also in the vesiculae seminales. The external genitalia and the secondary sexual characteristics were entirely of the male type. However, it appeared from the life history of this hermaphrodite that he was incapable of sexual intercourse, and that he had been feeble-minded from the age of six. According to Klebs's classification the proper denomination for such a case should be "*Pseudohermaphrodisismus masculinus internus*." The author, however, is of the opinion that both true and pseudohermaphroditism are different manifestations of one and the same primary deviation from the norm, affecting, in changing degrees, the whole generative system.

V. Keussler's paper has the double purpose of proposing a new classification of hermaphroditism and of showing that Steinach's doctrine of the determining influence of the interstitial gland of the testis ("puberty gland") upon the secondary sexual characteristics, is untenable. For such a purpose three different cases are considered. The first one, previously described by Brühl (*Diss. aus d. path. Inst., Freiburg i. B.*, 1892-4), was a child of two and one-half months with female external genitalia, vagina, and uterus of normal form and structure; a cord-like structure, morphologically similar to the Fallopian tube, was shown by Brühl to be without lumen. In the place of ovaries, testicles were found histologically identical with those of a normal child of the same age. No trace of the epididymis and vas deferens was seen in many serial sections. This case is against Steinach's theory, because no pathological change of the interstitial gland was found. According to the new classification it should be termed: *Pseudohermaphroditismus anatomicus (P.H.A.) certus masculinus genitalis subsidiarius internus et externus*. The second case was a twenty-year-old subject considered as a girl in spite of the fact that the body conformation was, on the whole, more that of a boy than that of a girl, and that the beard had

started growing two years before. The external genitalia consisted of a small and totally hypospadiac penis, so that the urinary meatus might have been mistaken for a short and narrow vulval slit. There were no testicles, and the two halves of the scrotum were so short and flabby as to look like the labia majora. The perineum was of the male type. The subject's voice had a peculiar tone intermediate between that of a man and that of a woman. An operation was performed during which the existence was ascertained of a testis with epididymis and of an oviduct with fimbria on the left side, of an oviduct with a body similar to an ovary on the right side. The left "testis" and right "ovary" were surgically removed and histologically investigated. It was thus proved that the left generative gland had really the structure of a testis, though without spermatozoa. Its interstitial tissue was richly developed. The right ovary-like body was, as a matter of fact, chiefly formed of interstitial cells, in the main similar to those of the normal ovary, but the apparent follicles passed through many transitional stages, into canaliculi in every way similar to the tubules of the left testis. In places, the interstitial tissue of this structurally rather undetermined body was so thickly arranged as to recall the picture of an endothelioma. v. Keussler, therefore, admits that this case could not be said to be quite against Steinach's theory, since the interstitial tissue was, at least on the right side, so highly developed and similar to that of the female endocrine gland as to overshadow the fact that on the other side the interstitial gland was of an undoubtedly male aspect, and that the hermaphrodite had external characteristics of a partially female type. According to the new classification this case should be termed *P.H.A. incertus subsidiarius int. et ext.* The third case of v. Keussler appears to be decidedly against Steinach's doctrine. The subject was apparently a "happily married woman" who went into a women's hospital because of a gonococcal urethritis. The body conformation could have been described as a mixture of male and female anatomical characteristics. The external genitalia were those of the female sex, but the normal vagina ended blindly in a sort of scar tissue. No trace of the uterus and adnexa were found. From both inguinal canals a testis-like body was removed. This was first investigated by Aschoff and then by v. Keussler, and found to be formed of tubules morphologically similar to those of a normal testis, but lined by a nondifferentiated epithelium which might have consisted of Sertoli's cells. In places the tubules had a very thin wall and were tightly arranged in nodular masses like adenomata. The interstitial tissue was not exceedingly developed and in every way similar to that of a normal testis. If Steinach's theory of the determining influence of the puberty gland were true, this interstitial tissue ought to have been like that of an ovary. From the point of view of the new classification, this case should be termed "*P.H.A. incertus subsidiarius int. et ext. extragenitalis ext. et psychicus.*"

In connection with the above observations and discussions, Poll's investigations on the testicles of two hybrids obtained from a peacock and a guinea-hen may be mentioned. The two birds were killed when four and five years old and termed Nos. 269 and 280. Each of them had two testicles, the left of which was larger than the right, but the left testis of the hybrid No. 280 was larger than that of the same side of the hybrid No. 269. A common characteristic of the four testes was the disappearance and degeneration of the seminiferous tubules, while the interstitial cells had multiplied to such an extent as to form nodules in every way similar to those of an "alveolar new growth." The author was not able to decide if these tumors of the interstitial tissue were really malignant in character, as no secondary nodules were found in other organs. However, he points out that the absence of metastases might have been due to the fact that the two hybrids were killed too soon, and that "disseminated tumors" of the abdominal cavity were observed by Ghigi (*Mem. d. r. Acc. sc. di Bologna*, 1911, VII, 331) in the only hybrid of the same sort which appears to have been previously, though incompletely, investigated. At any rate, and whatever the degree of malignancy of these tumors might have been, the facts observed point to the existence of an intimate relation between degeneration of a malformed germinative tissue and hyperplasia of the interstitial cells, both phenomena being probably connected with an original defect of zygotes due to the conjugation of heterogeneous gametes. This in the case of hybrids. As, however, similar facts have been described in the ootestis of human hermaphrodites, one feels justified in thinking that such deviations from the normal development might likewise be due to an original defect of the gametes or of the product of their fusion. [Da Fano.]

Leupold, E. INFLUENCE OF THE THYMUS ON THE DEVELOPMENT OF MALE GENERATIVE GLANDS. [*Beitr. z. path. Anat. u. z. allg. Path.*, 1920, LXVII, 472.]

The present paper is a continuation of the author's investigations on the relations between suprarenal glands and male generative glands (*Veröffentlichungen a. d. Geb. d. Kriegs-u. Konstitutionspath.*, Fisher, Jena, No. 4, 1920). As a conclusion to his previous and present researches he puts forward the following suggestions: The thymus appears to have a determining influence upon the development of the testes in extra-uterine life in general, and particularly upon its slow but steady growth during childhood and puberty. The size of both thymus and testes is constitutionally determined; these last, however, reach their constitutional dimensions and sexual maturity only during puberty provided that the thymus be unimpaired. But if the normal involution of the thymus is pathologically accelerated, the development of the testes is hindered even during childhood, and they may undergo a more or less marked process of atrophy. The further influence of the

thymus on the full maturation of the testes appears to manifest itself through the intermediary of the suprarenal glands. Indeed when these are either hypo- or hyper-plastic, the testes are likewise either smaller or larger than in normal conditions. If the thymus undergoes an involution too early, the suprarenal glands are insufficient *per se* to further the progressive development of the testes or to prevent their atrophy. On the other hand, in cases of persistence and hyperplasy of the thymus, this, provided the adrenals are normally developed, continues to influence the growth of the testes, which may reach uncommon dimensions and a very high degree of histological and functional maturity. However, this coöperation of thymus and adrenals in furthering or hindering the final development of the testes is not quite clear and needs further investigations. [Da Fano.]

Cutler, E. THE RELATION OF THE HYPOPHYSIS TO ANTIBODY PRODUCTION. [J. Exper. M., 1922, XXXV, 243. Med. Sc.]

After partial removal of the hypophysis cerebri, guinea-pigs were immunized with *B. typhosus* and were found to behave like the normal controls which had not been subjected to the operation. When previously immunized partial hypophysectomy had no influence on the continued production and persistence of typhoid agglutinins, hemagglutinins, or hemolysins. Feeding with hypophysis was also without influence. Total removal of the hypophysis proved fatal in 24 to 48 hours.

Jung. PREGNANCY AND HYPOPHYSEAL HYPERTROPHY. [Schweizerische medizinische Wochenschrift, January 19, 1922, LII, 3.]

The original discovery of this coincidence was made by Erdheim and Stumme, with the occasional supervision of acromegalic alterations in the women. There is abundant evidence of an antagonism between gonads and hypophysis. In gestation underfunction of the ovaries is associated with overfunction of the hypophysis. The increased size of the latter appears in radiograms through the expansion of the sella. Despite this enlargement it seldom leads to clinical manifestations of a severe character. In but two recorded cases did the hypertrophy cause symptoms of acromegaly or tumor in the sella. Petty symptoms pointing to the hypophysis are not so rare. The aim of the author is to add to the severe recorded cases another personal to him, which is strikingly like one of its predecessors. The woman was forty-five years old, seven months pregnant, with history of nine normal pregnancies in which naturally there were no visual disturbances. Ever since the last conception she has complained of progressive impairment of vision. On admission with this history an enlargement of the hypophysis was suspected and the X-ray showed the correctness of the assumption. The condition was so urgent from the standpoint of the prevention of blindness that abdominal hysterotomy was at once performed with tubal

sterilization. At the end of three days the patient could distinguish objects and individuals. Aside from the evidences of compression of the chiasm there were no other symptoms, hyperpituitarism being absent throughout in all of its manifestations. At the present time there is some improvement in visual acuity, but the fundal finds point to considerable permanent damage of both optic nerves. There is complete bitemporal age of both optic nerves. There is complete bitemporal hemianopsia with contraction of the visual fields to the fixation point. The author refers to Fehr's case in which pressure on the optic nerve by the pituitary, enlarging early in the second pregnancy, had caused bitemporal hemianopia which had persisted for ten years to date of writing, but with no signs of acromegaly, polyuria or glycosuria. Sella turcica was abnormally large. In his own case after the abortion, the rapid progress and severity of the visual disturbances and their immediate retrogression suggest that the pituitary must be the seat of a latent adenoma. Under the influence of the pregnancy the pituitary became congested, and exerted dangerous pressure on the optic nerve. Arresting the pregnancy relieved the congestion, and the adenoma subsided into its former latent phase.

Camus, J., Roussy, G., and Le Grand, A. DIABETES INSIPIDUS DUE TO LESION OF INFUNDIBULUM. [Compt. Rend. Soc. de Biol., LXXXVI, April 1, 1922, p. 719.]

A man was admitted for epileptiform attacks and transient palsies without disturbances of the reflexes. Since August, 1920, abundant polyuria without glycosuria. Early in January, 1921, he had paresis of upper limbs and violent headaches. He had encephalitic symptoms, convulsions, paralysis, with bradycardia and tuberculous arthritis of left knee. He vomited on January 15th, and died next day. The cerebrospinal fluid showed slight lymphocytosis and hyperalbuminosis (0.48): negative Wassermann and benzoïn test. Extract of bovine posterior pituitary lobe gave inconstant and temporary effect on the polyuria: antipyrines (3 grm. a day) and also novocaine (0.15 grm. a day) regularly diminished the amount of urine: lumbar puncture likewise (temporarily). Necropsy revealed an abscess in the sella turcica, of the size of a small hazel nut: it had entirely destroyed the pituitary (macroscopically and microscopically): the pituitary stalk was preserved. The infundibulum was not dilated, as to the naked eye the infundibular region appeared to be normal. But serial microscopical sections of the opto-peduncular region showed the presence of inflammatory lesions in the para-ventricular nuclei, infundibulum. The inflammatory lesions (of the hemorrhagic encephalitis) were symmetrical, and the interstitial tissue was edematous. The cellular lesions were also symmetrical, and were especially marked in the para-ventricular nuclei: they are less pronounced and more limited in a part of the special nucleus of the tuber and of the suprachiasmatic nucleus. In the tuber special nucleus

it is the larger cells that are affected: there are numerous elements with jagged borders and in a state of chromatolysis: there is also a moderate increase in the number of neuroglial cells. In the suprachiasmatic nucleus also only the largest cells are affected. In this case microscopical examination proved that the polyuria was due to the infundibular lesion and not to the destruction of the pituitary. The case, then, agrees with the experimental findings of Camus and Roussy, Houssay, Carula and Romana, Leschke, F. Bremer, and Pearce Bailey. In man, as in animals, diabetes is dependent on a lesion of the nuclei of the infundibulum and tuber cinereum and not of the pituitary body or its stalk. [Leonard J. Kidd, London, England.]

Hunter, Jr., J. W. ADENOMA OF PITUITARY. [Va. Med. Monthly, January 1922, XLVIII, No. 9. J. A. M. A.]

All of the five patients reported on by Hunter primarily complained of failing vision. In two cases there was a complete bitemporal hemianopsia for all of the colors; in one there was a greatly contracted field of vision for either eye with a bitemporal hemianopsia for the blue and red; in one there has been a gradual loss of sight, that of the left eye being entirely gone when seen and a temporal hemianopsia in the right, the blindness becoming total in time; and in one a complete blindness existed when seen. This emphasizes the need of mapping out the fields of vision in all doubtful cases. It is furthermore noted that in one case there was a distinct notch in the outer and upper quadrant of the fields of vision.

Keegan. SIGNS OF PITUITARY TUMOR. [Amer. Journ. Ophthal., November 1921. B. M. J.]

In this clinical paper the author discusses the disturbances of function of neighboring structures caused by pituitary tumor. Mainly ocular, they are summarized for the benefit of the ophthalmologist and rhinologist who may be first consulted for such disease, since the majority of early diagnostic signs of pituitary tumor relate to the eye and nose. The visual symptoms depend upon the forward intracranial extension and tumor pressure upon the optic chiasma, resulting in simple optic atrophy without choked disc, and bilateral hemianopsia, the color fields showing an earlier defect than the form fields. By first pressing on the under surface of the chiasma a visual field defect in both upper temporal quadrants is produced, further extension below the optic nerves resulting in a more complete temporal hemianopsia. In advanced cases of tumor with extensive intracranial infiltration oculomotor paralyses may result, and in some cases exophthalmos from involvement of the cavernous sinus. Anosmia may develop from destruction of the olfactory tracts, and pressure on the uncinate region may cause epileptiform attacks with a gustatory or olfactory aura. Epistaxis, intermittent discharge of mucus, and hypertrophy of lymphoid tissue are among

nasopharyngeal signs which, from nonrecognition of the underlying cause, may lead to ineffective nasal surgical interference.

Kraus, E. J. PANCREAS AND PITUITARY BODY. [Beitr. z. path. Anat. u. z. allg. Path., 1921, LXVIII, 258.]

By means of complete or almost complete extirpation of the pancreas it is possible to cause in cats changes of the pituitary body which do not essentially differ from those observed by the author in the same organ in cases of diabetes mellitus (Virchow's Arch. f. path. Anat., etc., 1920, CCXXVIII, 68). Such changes chiefly occur in the anterior lobe of the hypophysis, where an almost complete disappearance of the oxyntic cells can be observed. The surviving ones, which are irregularly distributed throughout the organ, appear smaller than in normal conditions, with a deformed cytoplasm and pycnotic nuclei. At the same time the average weight of the hypophysis is notably diminished. The alterations grow in proportion to the extension of the defect of pancreas, viz., of its islets. Hence the conclusion that the oxyntic cells of the pituitary body take an active part in the metabolism of sugar, and are functionally dependent upon the Langerhans islets.

After the extirpation of the pancreas in cats the pars intermedia and pars nervosa of the pituitary body likewise show changes of an atrophic character. The thyroid gland reacts to the removal of the pancreas, at first with an increased function, as proved by its increase in weight, but after a time it also undergoes a certain degree of atrophy. In the adrenals the lipoids disappear from the cortex, the chromaffinity from the medulla. As to the degenerative organs, an atrophy of the seminiferous tubules and of the interstitial cells of the testicle and ovary were observed in single cases. The pineal body likewise undergoes atrophy. The parathyroid glands appear to be the least affected. [Da Fona.]

Brunn. EFFECT OF HYPOPHYSIS EXTRACTS ON SECRETION OF URINE. [Zentral. f. in. Med., September 25, 1920, XLI, No. 39. J. A. M. A.]

Brunn states that while, on the basis of earlier animal experiments, it was reported that extracts of the hypophysis exert a diuretic effect on the secretion of urine, a large number of writers to-day agree that the subcutaneous injection of any of the commercial hypophysis preparations results in a checking of diuresis. Saxl and Brunn found that in from three to four hours after an injection of a hypophysis preparation a very small quantity of highly concentrated urine is secreted. In other trials an artificial polyuria was produced by allowing the subject to drink a liter of water; the half-hourly portions of urine were collected and the volume and specific gravity were determined. In from three to four hours the effect of the extract had worn off and copious diuresis set in, which corresponded to the normal results in experiments with water, only postponed, as it were, for four hours. The excretion of sodium chlorid was not affected, on the whole, by the extract. The checking of

diuresis was more marked the greater the original water diuresis and the less the sodium chlorid diuresis; in other words, the more dilute the urine and the lower the specific gravity. As throwing light on the possible mechanism of the effect of hypophysis extracts on diuresis, Brunn calls especial attention to the fact that during the checking of diuresis marked hydremia is noted. This seems to point to a blocking in the kidney and opposes the assumption that the tissues under the effect of the extract hold back the water, to a great extent, and cannot give it off into the blood stream.

Uhlenhuth, E. EXPERIMENTAL GIGANTISM PRODUCED BY FEEDING PITUITARY GLAND. [Proc. Soc. Exper. Biol. and Med., 1920, XVIII, 11. Med. Sc.]

By feeding the anterior lobe to salamanders specimens were produced which attained a growth much larger than any which have hitherto been seen. If the animals are fed on the posterior lobe, growth may be greatly retarded. Feeding produces both acceleration of growth and continuation beyond the specific size of the species.

Uhlenhuth, E. EXPERIMENTAL PRODUCTION OF GIGANTISM BY FEEDING THE ANTERIOR LOBE OF THE HYPOPHYSIS. [J. Gen. Physiol., 1921, III, 347. Med. Science.]

Metamorphosed salamanders (*Ambystoma opacum* and *tigrinum*) grew much more rapidly on anterior lobe of hypophysis than did controls on earthworms. Experimental giants were produced.

Bollack, J., and Nida. ADIPOSEO-GENITAL SYNDROME WITH OCULAR SYMPTOMS. [Presse Médicale, November 2, 1921, 880.]

The writers report a case of an adiposo-genital syndrome in a woman of twenty-seven. At the age of twenty a progressive adiposity appeared with menstrual disturbances but without any diabetes insipidus. Signs of increased intracranial tension a year ago: bilateral stasis of optic discs: the left eye blind from optic atrophy, with preservation of visual acuteness in the right eye in spite of narrowing of its nasal field. Radiography showed an apparently normal sella turcica: this appears to indicate an extra-sellar situation of the lesion. There is probably a tumor of the infundibulo-pituitary region with direct involvement of the optic chiasma. [Leonard J. Kidd, London, England.]

Hendry, J. CASE OF FRÖHLICH'S SYNDROME CAUSED BY INJURY TO SELLA TURCICA. [Glasgow Medical Journal, September 1921, XIV, No. 3.]

This casuistic article relates the history of a woman who fell, striking the back of her head. Following this she had almost complete loss of vision. This gradually returned though somewhat impaired. Amenorrhea developed, the patient became stout and signs of thyroid

insufficiency were apparent. Finally, a fairly well developed dystrophia adiposogenitalis, with lethargy and somnolence developed. The menstrual periods later returned but the flow was slight. The roentgen ray showed a fracture of the base of the skull involving the sella turcica. The patient could ingest 300 gm. glucose without showing any glycosuria. Her temperature was steadily subnormal and her blood pressure low (105 mm. Hg). Extract of the anterior lobe of the pituitary was administered with slow improvement following.

Grafe, E. ADIPOSIS DOLOROSA. [Munch. med. Woch., March 19, 1920.]

A clinical history of a girl who at the practically unrecorded early age of fourteen began to show signs of this syndrome. One case reported by White began at an early age also, *i.e.*, earlier than thirty. The development of the syndromes were also unusual. The deposits of fat are usually tender and spontaneous neuralgic pains are slight and occasional. In this girl there was scarcely any pain on pressure, but spontaneous burning sensations and subjective sense of great tension was present. Grafe thinks that the cause of the pains in his patient was accumulation of water in the fatty tissue, and perhaps in the musculature also. This alone would explain the intermittent swelling up of the deposits of fat and their subsidence at certain times. The vegetative control of the water tissue was evidently involved in some manner.

Kaufmann, F. ADIPOSIS DOLOROSA. [Schw. Arch. f. Neur. u. Psych., IX, 108.]

One of two cases of Dercum's disease manifested a strong tendency to hemorrhage in the form of hematemesis. No changes could be found in the hypophysis or thyroid. Delay in coagulation of the blood was characteristic of both cases. Hypothyroidism was suspected because of increase of carbohydrate tolerance, lowering of energy transformation, and in one patient the lessening of the fever reaction. Thyroid treatment brought about improvement in the psychic activity as well as in the general condition.

McKinlay, C. A. POSTERIOR LOBE OF PITUITARY AND METABOLISM. [Arch. of Internal Med., December 1921, XXVIII, 6.]

From a series of experimental administration of the posterior lobe of the pituitary this author advances the hypothesis that the average, *i.e.*, so-called normal person, responds quite constantly with increased basal metabolism following the subcutaneous injection of pituitary extract. In hypothyroidism, as judged from a small group of individuals, the basal metabolism was diminished rather than increased. This suggests to the author that pituitary extract is effective in accelerating heat production only in the presence of a normally functioning thyroid gland. In four cases with reduced basal metabolism without myxedema and the influence of endocrine glands other than the thyroid was

probable, a positive response to pituitary extract was present. In a group of healthy individuals following the subcutaneous injection of pituitary extract one week after an injection of thyroxin the resultant acceleration of the "basal metabolism" was interpreted as a synergistic action between the pituitary extract and the thyroxin.

Houssay and Hug. PITUITARY TREATMENT OF POLYURIA. [Revista d. 1. Asoc. Méd. Argentina, July 1921, XXXIV, 201.]

Injection of pituitary extract usually increases diuresis in normal dogs, or in those with cerebral polyuria, while it reduces diuresis in rabbits. It does not modify diuresis in the average human, but reduces it in the polyuric. Houssay and Hug recall that true endocrine action is uniform; removal of the thyroid reduces metabolism, and removal of the pancreas induces glycosuria in all species. The chief value of this short paper is to reemphasize the obvious, constantly overlooked by most of the endocrinological "investigators," that the function of an organ cannot be deduced by the pharmacodynamics of its extract (any more than the value of an American beauty can be judged by a prick of its thorns [Ed.]).

II. SENSORI-MOTOR NEUROLOGY.

1. CRANIAL NERVES.

Hobday, Frederick. VOCAL CORD PARESIS IN THE HORSE. [Journal of Laryngology and Otology, September, 1921].

This is a report upon an operation which is employed frequently for the alleviation of whistling or roaring in the horse—a condition due to paralysis of the left vocal cord. The method may be applicable to similar conditions in man, he argues. Especially in cases of bilateral abductor paralysis causing dyspnea and involving the risk of asphyxia and possibly necessitating the wearing of a tracheotomy tube, the operation of stripping the ventricle may prove useful. In the case of the horse an incision is made through the crico-thyroid membrane and the edge of the left vocal cord is incised along its whole length. The finger is then inserted under the mucosa, which is thus carefully stripped from the whole of the ventricular sac. The mucosa is then incised along the edge of the false vocal cord and the separated mucosal flap removed. The ventricle on the right side is similarly denuded of mucosa and the operation is complete. No suturing is done. The external wound heals in ten days and the animal is kept at complete rest for a month. It is then given slow walking exercises and tested at a gallop after three months.

Stevens, C. W. TRIGEMINAL NEURALGIA. [Arch. Ophth., March 1920.]

In view of the poor results from the surgical treatment of facial neuralgia, the author argues that more research should be made to determine the cause, then the old surgical experiments might well be abandoned. A clergyman, aged twenty-nine years, suffered every two months from violent pain in the orbit, supraorbital region and parietal eminence. There were at times loss of consciousness, epileptiform seizures and mental derangement. His vision had been corrected for hypermetropia and astigmatism. Further examination revealed 2° of esophoria and with the clinoscope a declination of + 2° in the right and + 5° in the left eye had been found. For clear vision it is necessary to keep the eyes vertical and this demands the assistance of the facial muscles. Hence, in this patient both brows were strongly compressed, the left more than the right. There was a deep, depressed, vertical ridge between the eyebrows and prominent transverse wrinkles on the forehead. There was spasmodic contraction of the whole face. Three operations were performed to correct the declination in both eyes, resulting in complete cure of the neuralgia. The cause of the neuralgia, he reasoned, was the abnormal pressure of the facial muscles on the supratrochlear and supraorbital branches of the ophthalmic division of the fifth cranial nerve.

Teale, F. H., and Embleton, D. THE PATHS OF SPREAD OF BACTERIAL EXOTOXINS, WITH SPECIAL REFERENCE TO TETANUS TOXIN. [J. Path. and Bacteriol., 1919, XXIII, 50. Med. Sc.]

This paper records the results of experiments in which animals were inoculated subcutaneously, intravenously or intraneurally with tetanus toxin or emulsions of cultures. Although they confirm the older view that tetanus toxin ascends by way of the axis cylinders, they show that it also passes to a great extent to the cord by the perineural lymphatics. If these paths are obstructed the occurrence of tetanus may be prevented. By injection of tetanus toxin directly into the blood-stream the authors found that in the space of minutes it had leaked into the peripheral lymphatics to appear in the thoracic duct. The toxin, however, does not pass from the blood into the cerebrospinal fluid, the capillaries of the central nervous system and choroid plexus having apparently a selective function which does not allow the toxin to pass through the posterior root ganglia to the cord, although colloidal dyes are apparently stopped in their course along afferent nerves. Tetanus toxin behaves in the same way. As is well known, iodine mixed with tetanus toxin renders the latter nontoxic by the subcutaneous route. The authors find, however, that when injected intracerebrally typical cerebral tetanus is produced. With regard to the site of action of tetanus antitoxin they consider that this is in the circulating blood or at the seat of production of the toxin only, as it cannot be demonstrated to have entered the central nervous system either by way of the blood-vessels, neural

lymphatics, or actual nerve substance. Tetanus toxin already fixed in the central nervous tissue is beyond the reach of antitetanic serum administered therapeutically.

Hays, Harold. HYPERTENSION AND HYPOTENSION OF THE MEMBRANA TYMPANI AND DEAFNESS AND TINNITUS. [New York State Medical Journal, June 1920.]

By hypertension of the ear drum one means that the drum membrane is more rigidly held in place than it should be. By hypotension of the drum one means that the drum is more flaccid. A certain poutiness may be present so much so that one wonders if there is not something in the middle ear which presses it out. In certain patients there is a combined condition of drum hypertension and hypotension. One sees a combination of the deeply indented drum with some relaxation in parts.

The causes of these conditions are, first, improper blowing of the nose which may cause trouble through a patulous tube. In these instances every bit of air which enters the nasopharynx enters the Eustachian tube and presses out the drum. The second class of cases is due to improper blowing through partially stenosed tube. When the nose is blown too forcibly, a certain amount of air gains entrance into the middle ear under considerable pressure. The tube closes. Whether the drum first becomes distended or not depends upon how much air escapes, how rapidly the contained air is absorbed and with what amount of force it reaches the membranous tympani. A second cause is frequent ear aches in children. Too little attention is paid to them, except at the time of the acute condition. A third cause is the neglect of discharging ears. Most otologists are careful about the treatment of ears while they are discharging but pay very little attention after the ear ceases to discharge. A suppurative ear is not cured until the hearing is properly restored and the sooner the child's parents are impressed with this fact the better off the child will be. Fourth, are adhesions in the fossa of Rosenmüller. The ordinary examination of the rhinopharynx with the mirror will not reveal the adhesions, so a more careful examination has to be made with the author's pharyngoscope or with the nasopharyngoscope of Holmes. Then one will often see fine bands in this fossa which cause the trouble. The result is that the tube is either held open too widely, causing a hypotension or else the muscles are held in the wrong position causing a hypertension of the drum. Fifth are polypoid posterior tips of the inferior turbinates. These are not as often recognized as they should be. They are easily seen on proper examination and are often of sufficient size to block the tubal orifice. Sixth, one must consider diseased teeth, or buried molars. These are etiological factors of as much importance as tonsils and adenoids and deflected septum, and are a source of continuous irritation to the nasopharynx.

The diagnosis is made by the examination of the Eustachian tube and the examination of the drum by means of an otoscope. The ex-

amination should be directed towards the tubal orifice where various pathological conditions are readily seen which result in either of the conditions, hypertension or hypotension. Secondary to these are the conditions within the tube. One should also notice the condition of the drum by noticing its vibratory excursions. One should also pay great attention to the variations in tubal patency because some tubes are exceedingly small while others are very large, either one of which may result in deafness. In noting tubal patency, one must consider the various sounds that are transmitted to his ear through the sounding tube. On gentle inflation one may hear crackling sounds or gurgling sounds or whistling sounds or a sucking sound or a mucoid sound, each one of which is significant in itself and should be taken gravely into consideration.

The otoscopic diagnosis is made possible by making use of the electric otoscope. One will be able to tell by the vibrations of the drum whether the drum is too tensely drawn or else too relaxed but one needs considerable experience in making this observation.

Treatment should be directed to the prevention of the conditions which includes the proper hygiene of the nose and throat and the removal of all pathological conditions therein which may cause an irritation in the Eustachian tube. The treatment of the ears will differ in cases of hypertension and hypotension. In the former, diseased conditions in the Eustachian tube must invariably be overcome, usually by the dilatation of the tube by Yankauer applicators, sounds and bougies. The technic to this can easily be mastered. After this is done one must pay direct attention to the condition of the mucosa of the Eustachian orifices and treat it accordingly. When hypotension is present catheterization may do a great deal of harm. One may use a mild Politzerization but every attempt should be made to tighten up the drum. This may be accomplished by continuous vibration of the drum or by the application of cantharides collodion as suggested by Heath of London. Where both hypotension and hypertension exist in the same ear, it is quite difficult to outline any definite line of treatment. Each case must be decided on its own merits. [Author's abstract.]

Retjo, A. TUNING-FORK TESTS. [Laryngoscope, July 1920.]

This clinical experimental paper discusses what the tuning-fork tests teach relative to localization. The end-organ of the cochlea has only one stimulus, viz., the wave-like motion induced in the labyrinthine fluid. In the case of air-conduction the perception of the lower and higher tones takes place with the help of the conducting apparatus. The only difference is that for perception of lower tones the transforming action of the drum and ossicles is necessary, whereas for the higher tones it is quite enough if the two windows function normally. In bone-conduction there must arise the same wave-like perilymph motion which is produced in the case of air-conduction, because it is the only

stimulus for the auditory nerve. This motion, directed towards the round window, arises in the labyrinthine fluid through the molecular vibration of the skull bones, but simultaneously there is produced another motion directed towards the oval window. As only the first of these motions is necessary for perception, in case of bone-conduction, only the functioning of the round window is required. If neither of the windows is elastic no wave-like motion can arise in the fluid and there is neither stimulus nor perception. If the lower tones are normal and the higher tones shortened in the case of air-conduction, the disease is in the perception apparatus. If the higher tones are normal and the lower tones are shortened, the disease is in the transforming part of the conducting apparatus. Shortening of the lower, as well as the higher, tones gives no definite information as to the local diagnosis and in this case several tuning-forks should be used. In regard to bone-conduction the shortening found in elderly persons is probably due to rigidity of the membrane of the round window and not to senile involution of the cochlear nerve. In middle ear inflammation the oval window usually shares with the ossicles and tympanic membrane in the swelling and immobilizing of the mucosa and there is lengthening of the bone-conduction. But if the round window is also involved, bone-conduction will be shortened and the perception of higher tones by air considerably diminished. In otosclerosis the shortened bone-conduction is probably due to ossification of the structures in the vicinity of the oval window and even those extending as far as the round window.

Love, James Kerr. ORIGIN OF CONGENITAL DEAFNESS. [Journ. Laryng., Rhin., Otology, September 1920.]

Sporadic congenital deafness is identical with true hereditary deafness obeying Mendelian characters. Six possible unions between the deaf and hearing in the human family are posited: (1) A hearing man, whose parents heard and did not carry hereditary deafness, marries a similar woman. No deaf children can result. Ninety per cent of human marriages are of this type. (2) A deaf man, whose parents are hereditarily deaf, marries a similar woman. All their children will be deaf. (3) A pure hearing person marries a pure deaf-mute and all the children hear, but several of the grandchildren are deaf. (4) A hearing man carrying deafness marries a hearing wife carrying deafness and both deaf and hearing children follow. Deaf children must follow if the family be large. (5) A hearing man carrying deafness marries a pure hearing woman and no deafness will result. But half of the children will carry deafness and if any of these wander into classes (2), (3), (4) or (6), deaf children will result. (6) A hearing man carrying deafness marries a pure deaf woman. Half the children are deaf and all the children carry deafness. This is a common type of marriage among the deaf. The six classes agree with the Mendelian phenomena, as displayed by the crossing of tall and short peas and the

subsequent self-fertilization of the resulting hybrids. Sporadic congenital deafness, according to the above view, is due to the meeting of two heterozygotes—hearing hybrids carrying deafness.

Fraser, J. S. PATHOLOGICAL AND CLINICAL ASPECTS OF DEAF-MUTISM.
[*Jour. Laryngology and Otology*, January-March 1922.]

The author gives a detailed account of the examination he has made of 140 deaf-mute children. He also compares his findings with the literature as to the pathology and possible etiology of the condition. He speaks of the impossibility of obtaining sufficiently extensive or accurate clinical data since the condition may develop in its obvious form when the developmental defect has long been in existence. He adopts a more scientific classification than the division into congenital and acquired deaf-mutism, dividing the cases into those due to an error in development and those due to trauma or to inflammatory conditions.

In the first class there may be endemic or cretinic deafness where the latter is usually associated with feeble mentality or idiocy so that even a moderate degree of anatomical deafness would result in deaf-mutism. The anatomical changes are myxedematous, thickening of middle ear tissues, intrusion of connective tissue, fat cells, bony tissue with general otosclerosis and almost complete bony obliteration of the tympanum. There may have been otitis media. At any rate, contrary to the cases of sporadic congenital deafness, it is chiefly the middle ear that is affected. Opinion differs as to whether the changes are constitutional in origin or due to intrauterine infection or toxic disturbance. In sporadic congenital deafness the membranous labyrinth shows marked changes which causes collapse or dilatation of the cochlear duct, the cochlea showing also a lower phylogenetic development. The vestibular apparatus also shows interference with its development. Here hereditary weakness of the ear may show itself in atrophic degenerative processes which may begin in intrauterine life or later. They seem to be due to lack of nervous energy to bear the strain of activity. The condition has been attributed to intrauterine meningitis, which the author disputes because the membranous labyrinth is not developmentally in connection with the central nervous system at this time and the changes themselves are not those which would be expected. Otosclerosis is probably present in varying degrees and forms a transition between the early defect and the later obvious degree of deafness. It may be due to determinants in the germinal cells of the parents which then owe their further development to extraneous causes, age, critical period, middle ear disease. The otosclerosis is probably only one manifestation of a larger process.

Acquired deaf-mutism is easier to determine as to its origin since its pathology is that of labyrinthitis of the intrauterine or post-fetal life, the former due to meningitis, the latter to traumatism, invasion from otitis media or purulent leptomeningitis passing along the eighth nerve or the aqueduct of the cochlea. About 4 per cent of the cases of acquired

deaf-mutism have a traumatic origin. The growth of new bone and of connective tissue after fracture of the petrous bone causes filling up of the perilymphatic space, dilatation of the cochlear duct and atrophic degeneration of the neuroepithelium and spiral ganglion. Otitis media may produce deaf-mutism if it is sufficient to close both labyrinth windows. There may be great destruction of the middle ear present with the otitis or scarlatina or of measles while tuberculous middle-ear disease may also produce deaf-mutism. Syphilitic deafness may be due to a labyrinthitis secondary to the meningitis or to the nonresistance in syphilitic children to the invasion of middle ear inflammation. Purulent meningitis may be intrauterine or a result of epidemic meningitis or other infectious disease. Here there is marked invasion and destruction of the labyrinth. Post-fetal meningitis, chiefly due to epidemic meningitis, is the most frequent cause of acquired deaf-mutism. The writer discusses the particular changes occurring with the meningitis of various infectious diseases. He cites the examination of a number of ears of syphilitic infants which seemed to show that "congenital" syphilitic deafness is due to an extension of the syphilitic process along the acoustic nerve. Consanguinity of parents appears in about 12 per cent of deaf-mutes. Direct heredity of deaf-mutism is rare except when both parents are deaf-mutes. Comparison of statistics gives a rate of 52 congenital cases, 43 acquired and 5 doubtful; also of 36 in 100 cases due to epidemic meningitis, 16 to scarlet fever, 10 to measles, 10 to pneumonia, 11 to syphilis, 4 to trauma, 3 each to whooping-cough, mumps and typhoid fever, 2 each to influenza and pneumonia. Congenital syphilis, even with the aid of the Wassermann test, is not easy of discovery as a cause. As the figures stand, 2.5 to 18.6 per cent of cases may be so attributed. [Author's abstract condensed.]

Olmsted, J. M. D. DEVELOPMENT OF TASTE BUDS. [Journ. Compar. Neurol., June 1920.]

The influence of one organ upon the development of another is a fundamental problem in morphogenesis. The differentiation of specialized organs, such as the transformation of epithelial cells into taste buds, has been held to be due to the growth of the appropriate nerve into the region concerned. Additional evidence that the presence of the nerve is the formative influence in the development of taste buds is thought to be brought. The material experimented on was supplied by the barbels of the catfish, *Amiurus nebulosus*, the ends of which were cut off and then the process of regeneration carefully followed through every step. It was found that in all stages of regenerating ends of barbels the nerve and cartilage extend practically the complete distance from the old stump to the basement membrane of the epidermis at the very tip. Short regenerated pieces show no trace of taste buds. The formation of dermal papillæ, the invariable forerunners of taste buds, takes place at the base of longer regenerated pieces, as if the germinated layer of the epidermis

were indented by the growth into it of a small branch of the nerve trunk. Each papilla is filled with a small bundle of nerve fibers which stand out from the nerve trunk like a small button, causing this indentation of the germinative layer. Later stages show the presence of fully developed taste buds along the whole length of the regenerated end, mainly concentrated, however, along the edge nearest the nerve. Since taste buds degenerate in a barbel whose nerve is cut and reappear when the nerve regenerates and since the nerve appears in the appropriate region before there is any evidence of a developing taste bud, the presence of the nerve may be said to be the causative factor in the formation of taste buds.

De Martel. ACOUSTIC NERVE TUMORS. [Bull. Mém. Soc. de Chir. de Paris, November 1920.]

This surgical technic paper describes his operation for tumors of the acoustic nerve. The results of operations upon these cases heretofore have been so bad that the physician who sees the cases is chary of handing them on to the surgeon. Three successes out of five attempts has been the author's record and he claims that these are the first cases of recovery after operation published in France. De Martel uses Cushing's technic with two variations. He has the patient in the sitting posture astride a chair, with his forehead resting on the arm, and infiltrates with novocain instead of giving a general anesthetic. These two points are of value, as hemorrhage is less and the patient is able to aid the surgeon by moving the head. These tumors are usually small, occupy the cerebellopontine angle, and deafness is an early symptom.

Lozano. POSTDIPHTHERIC PARALYSIS. [Arch. Esp. d. Ped., April 1920, IV, No. 4.]

The diphtheria in the boy of three had not been recognized or treated, but the pains, head drop and generalized paralysis about a month after the febrile sore throat improved rapidly under antitoxin treatment. He was given a total of 20,000 units, 5,000 units on alternate days, and by the end of the month the only trace left of the paralysis was the loss of the knee jerk and an almost imperceptible difference in the use of the right leg.

Hayes, Harold M. FIVE HUNDRED CASES OF PROGRESSIVE DEAFNESS. [Annals of Otology, Rhinology, Laryngology, December 1921.]

The author analyzes over five hundred cases of progressive deafness which have occurred in his private practice and although he goes into no specific details, he comes to a very definite conclusion. He claims that the more careful inspection of the nasopharynx, especially the regions of the Eustachian tubes, by means of the Holmes' nasopharyngoscope, has allowed of a more careful study of these conditions. He does not feel that heredity plays a great part in these cases, except

in so far that there is an hereditary predisposition to a weakness of the ear mechanism. The diseases of childhood play a great part in altering the mechanism of the middle ear, especially in causing progressive deafness. There are two classes of cases which deserve particular attention. Patients who have had exanthematous diseases and those who have had recurrent colds which are so often associated with diseased tonsils and adenoids. Many of these children do not complain of their hearing until after the age of puberty, although many of the cases had nose and throat abnormalities, some of which had been corrected. He regrets to say that the correction of the nose and throat trouble has not, by any means, improved the hearing in many of these cases. He divides his cases according to a clinicopathologic classification as follows: 1. Retracted drum with stenosed tube. 2. Retracted drum with open tube. 3. Slightly retracted drum with a tube which intermittently opens and closes. 4. Slightly retracted drum which on vibratory massage shows only slight loss of motion. 5. The relaxed ear drum associated or unassociated with a retracted drum. 6. Rare cases such as otosclerosis and nerve deafness.

In speaking of prognosis, he divides his cases into two classes: patients who are moderately deaf and in whom there is some hope of improvement and cases that are hopelessly deaf from a medical point of view. For the latter, he believes that mental reconstruction is of the utmost importance and is the duty of the otologist. He feels that the prognostication of the future will depend on how much educational propaganda can be spread about so that the proper preventive measures in early childhood will be used. Among suggestions in treatment he considers the following: (a) Attention to the pathological condition of the nasopharynx which exercises any influence on the Eustachian tube. Tubal patency is of the utmost importance. (b) The use of the high frequency current in the nasopharynx and (c) finally, the proper dilatation of the tubes by various bougies and sounds. [Author's abstract.]

Benon and Kerbrat. POSTTRAUMATIC PERSISTING HEADACHE. [Schweiz. Archiv. f. Neurol. u. Psychiatrie, 1921, VIII, No. 2.]

A clinical paper dealing with an obstinate cephalalgic syndrome on the basis of the analysis of three cases.

Busacchi, P. NERVOUS MANIFESTATIONS AFTER DIPHTHERIA. [Riv. di Clin. Ped., March 1921, XIX, No. 3. J. A. M. A.]

Busacchi refers to the involuntary movements associated with the volitional movements in children with paralysis after diphtheria. In some this synkinesia is restricted to the face, and it may precede the paralysis. Sometimes the movements occur in connection with pronouncing certain letters. The child's character may change, fluctuating between whining peevishness and wild glee, the face growing very red at times. Others present motor phenomena suggesting chorea by their

sequence and automatism, but not so irregular. The raising of the eyebrows, frowning, wrinkling the eyelids, etc., occur rhythmically and symmetrically.

de Parrel, G. SAVING THE REMNANTS OF HEARING. [Brazil Medico, November 12, 1921, II, No. 18. J. A. M. A.]

De Parrel expatiates on the importance of training the ear to utilize the last remnants of hearing, and by functional exercises improve the function. Sound waves and other stimuli, means to improve the blood supply, and mobilization of the muscles of the internal ear all aid in the reëducation of the hearing, and he describes the procedures best adapted for these purposes. With absolute deafness, of syphilitic, meningitic or traumatic or central origin, it is not worth while to attempt to retain the hearing, and there is no recourse but to learn lip reading. But it is astonishing what progress can be made by training the attention, training in listening, and arresting the further progress of the ear disease, as he explains in detail.

Young, Gavin. THE VESTIBULAR REACTIONS IN DEAF-MUTES. [Journal of Laryngology, November 1921.]

The writer has investigated the labyrinthine capacity in a series of deaf-mutes. Two tests were employed: (1) syringing the ears alternately with hot and cold water, either until nystagmus was elicited or till two minutes had elapsed, and (2) rotation of the subject ten times to each side. Three classes of deaf-mutes were chosen. The first were cases of hereditary deaf-mutism, *i.e.*, children who were born deaf and who had a well-established family history of deaf-mutism. In the second class were children who had been born hearing but had sustained later in childhood an attack of cerebrospinal meningitis which had resulted in total deafness. Congenital syphilis was the pathogenetic agent in the third group. The Wassermann reaction was positive in each case, and the children each bore one or more syphilitic stigmata, peg-top teeth, interstitial keratitis, etc.

This clinical study bore out fully what is already known pathologically of this not very fully investigated subject. That is to say, the hereditary deaf-mutes suffer from some developmental error in their hearing, as opposed to the balancing apparatus of the internal ear. The cochlea therefore functions defectively. The semicircular canal system, however, is not affected by this condition, and as might be expected, the subjects of the first group gave reactions to the tests which were normal or nearly normal. The members of the second class, those rendered deaf by cerebrospinal meningitis, failed entirely to respond to any stimulus of the labyrinth. This also is to be looked for, when it is remembered that the whole thickness of the auditory nerve is involved, whether in an actual neuritis or in the nipping caused by the formation of post-meningitic adhesions. The results in the third show that congenital

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syphilis may destroy the whole labyrinth or only a part of it, or attack the whole nerve as in group two, or the labyrinth may be avoided by the process altogether. In eight cases examined in this group, three gave normal reactions while five gave no response. [Author's abstract.]

Labbé, R. DIPHTHERIC PARALYSIS AND SEROTHERAPY. [Archives de Médecine des Enfants, October 1921, XXIV, No. 10.]

Six cases of late diphtheric paralysis are here described as treated by antitoxin. The children were from three to thirteen years old and the paralysis had developed a month to six weeks after the beginning of the sore throat. They were all benefited by the systematic use of the antitoxin and in one case with a residual soft palate palsy of four years' standing there was complete restitution of function.

Kolodziej, H. TREATMENT OF TRIGEMINAL NEURALGIA. [Medizinische Klinik, July 17, 1921, XVII, No. 29.]

Complete cure is here recorded following two injections of 1 c.c. of 70 per cent alcohol into the ganglion.

Shea, J. J. VERTIGO. [Jl. Tenn. State Med. Assoc., August 1921.]

Vertigo is a subjective sensation of a disrupted relationship of one's own body to surrounding objects in space. In other words, it is a disturbance of one's equilibrium. Hence a study of vertigo must be based upon a study of the method of equilibrium. All conscious sensations are cerebral. The brain perceives the combined information from the muscle joint sense, the sight and the static-kinetic sense and thus becomes conscious of one's relationship to the object in space about it. The tabetic is deprived of the muscle joint information and staggers, but is seldom dizzy. The sight by the power of fixation gives information as to the relative position of objects about us, and when there is an imbalance of the external muscles of the eyes the brain is confused because the information from the two eyes do not coincide. Here I wish to correct an erroneous impression which has gained cognizance in medicine, that the eye is often a cause of vertigo. Only when the external muscles are not working in symmetry does it produce vertigo.

This then leaves the static-kinetic sense as the main source of knowledge of equilibrium; that is, static-kinetic is truly a special sense and may be proven by its conforming to the requirements of the special sense, *i.e.*, an organ for the reception of the stimulant, a special nerve for the transmission of the stimulant and a localized area in the cortex for the perception of the stimulant. The labyrinth of the ear is the organ, the vestibular branch of the eighth cranial nerve is the special nerve and the temperoparietal is the localized area of cortex. The labyrinth may be considered to be composed of three semicircular canals and two reservoirs. Into the lumen of these canals are sensitive nerve endings which will interpret the movements of the body with regard to space.

Inasmuch as there is a special organ, nerve and brain area for this sense, it is only through the study of this organ and its nerve tracts that a proper diagnosis of the cause of the vertigo may be reached. We all know that an intoxication, either intestinal or otherwise will produce vertigo, but how? By its action on this special organ and its nerve tracts. Vision may be reduced by intoxication, but only by the action of the toxin on the organ of sight or its nerve pathways. If we consider the information from each labyrinth as reins upon whose tension we are guided, it is readily seen that when there is an unequal pull it will spin us instead of driving us straight. In time we can accommodate ourselves to this information and go steady, but as soon as there is another upset we are confused by the new imbalance and have the subjective sensation of vertigo.

Vertigo may then be of two great types: The first where the system of equilibrium is intact, but their correct information is disturbed due to the action of toxins on the organ or its nerve pathways. This toxin may be temporary or permanent—alcohol or the virus of syphilis. In this class the Bárány tests will show the pathways open, but some increase or lessening of the normal reactions or an unequal information from the two sides. The second type is where the Bárány tests show a blocking or absence of passage through one or all of the tracts and the vertigo is due to an organic lesion. No one has a right to-day to presume that so serious a symptom as vertigo is caused by any one factor, without first having a Bárány test correctly made and interpreted upon by his patient. [Author's abstract.]

Trogu, G. ANESTHESIA OF TRIGEMINAL. [Policlinico, March 1921, XXVIII, No. 13. J. A. M. A.]

Trogu emphasizes the advantage of having the patient conscious in operations on the jaw, as aspiration of secretions can thus be averted. He has found injection of alcohol into the gasserian ganglion the most effectual and convenient method for local anesthesia, and also for treatment of trigeminal neuralgia, and reports two cancer cases and two neuralgia cases to demonstrate the superiority of this technic, by Härtel's method.

Marie and Mathieu. DIPHTHERIC PARALYSIS IN ADULTS. [Bull. d. l. Soc. Méd. des. Hôp., December 2, 1921, XLV, No. 35.]

A clinical report upon two cases in adult males of eighteen and thirty-four years, respectively. The paralysis was spinal in type, and there was disturbance in swallowing, speech, and accommodation, and partial loss of power of arms and legs were present. Diphtheric paralysis in adults is rare. Two or three cases a year at the Salpêtrière Service has been the average. These cases came in two weeks. Ramond stated that 18 per cent of the soldiers were paralyzed in an epidemic of diphtheria in one regiment until injecting 5 or 10 c.c. of the anti-

toxin intraspinally was done, as a supplementary measure. The authors point out that the diphtheria toxin merely induces a peripheral neuritis but, when very virulent, the spinal cord may be involved.

Magnus. THE TREATMENT OF TRIGEMINAL NEURALGIA. [Norsk. Mag. for Laegevidenskaben, June 1921. B. M. J.]

This author summarizes his review of this subject in the two following statements: (1) There are only two effective methods for the treatment of trigeminal neuralgia—jections of alcohol into the branches of the nerve, and excision of the Gasserian ganglion or its pontine root; (2) peripheral resections of the nerve are obsolete because their place can be taken by the far simpler measure of alcohol injection. The author has performed peripheral resections of the nerve in 29 cases, in all of which a relapse occurred in twelve to eighteen months. He has given 248 injections of alcohol into various branches of the nerve in 118 cases and in one of these cases the patient was free from pain for eight years. Four patients were free from pain for five years, and the average duration of freedom from pain was twelve to eighteen months. There was little difference in the effects of peripheral or more central injections. After 211 of the 248 injections the pain disappeared at once; 37 injections failed of their object. The author has never injected alcohol into the Gasserian ganglion, and he justifies his opposition to this procedure by references to complications, some fatal, which this method has provoked. He is far better pleased with operative removal of the Gasserian ganglion or its pontine root, and in not one of the 31 cases which he has operated on has he seen paralysis of the ocular muscles, and only in one case did transitory facial paralysis occur.

Hallock, W. H. POSTDIPHTHERITIC PARALYSIS. [N. Y. Med. Jour., April 1921, CXIII, No. 12. J. A. M. A.]

The first sequel to appear in Hallock's case occurring in a man, aged fifty, did so promptly, and consisted of a paralysis of the pharyngeal muscles which persisted for a week. Weight and strength gradually returned and in one month the patient was able to do light work about the farm. Three months after the acute illness it was first noticed that the wrists and ankles were becoming weak. This weakness gradually involved the arms and legs until the patient was again rendered helpless, being unable to help himself in any way. There was a complete loss of use of the flexor and extensor muscles of the fingers and toes, wrists and ankles, with partial loss of voluntary motion at the elbows and knees. All of the muscles moving the shoulders and hips functioned, but weakly. There was a beginning atrophy of the muscles most extensively involved, especially the dorsal interossei of the hands. The abdominal muscles of the left side were flaccid and could not voluntarily be tightened, while those of the right side were definitely weakened. The anal sphincter functioned normally, while the vesical sphincter was slightly involved. All tendon reflexes usually elicited from the extremi-

ties were unobtainable, even by reinforcement. The left abdominal reflex was absent; the right, weak. The cremasteric was present bilaterally. Neither a positive nor a negative Babinski was obtained. Three nose and throat cultures and smears at three day intervals were negative for the diphtheria bacillus. Strychnin was at once started by hypodermic injection and continued to beginning toxicity and maintained just below the danger point. Massage and passive motion were employed daily. No diphtheria antitoxin was administered at any time. In ten days the patient was able to sit up in bed and make coarse movements of the arms and legs, but the hands and feet were useless. Sixteen days from admission, the patient was first able to grasp a fork in his hand and bear weight on his feet without toppling over. On the twenty-third day symptoms of strychnin poisoning appeared. On the twenty-ninth day crutches were used for the first time. On the thirty-fourth day the patient first walked without crutches. Five months after admission the patient was in excellent condition.

Ardenne. THE WASSERMANN REACTION IN DEAF-MUTES. [Rev. de lar., d'otol. et de rhinol., May 31, 1921.]

In 23 cases of deaf-mutism the W. R. showed the following: In one group of 11 children the deafness was congenital. In this group the reaction was positive in 8 and negative in 3. A second group consisted of 12 cases in which the deafness could be explained by a lesion of the middle ear or by a disease in infancy, such as meningitis, or cases in which the cause of deafness was unknown. In this group the Wassermann reaction was more or less positive in 5 and negative in 7. Although definite conclusions could not be drawn from so small a number of cases, Ardenne suggests that a systematic study of the Wassermann reaction in deaf-mutism would throw light upon the etiology of this infirmity.

Lemere, H. B. TIC DOULOUREUX AND LATENT MAXILLITIS. [Nebraska State Journal, 1921.]

This author summarizes this interesting research as follows: Treatment of the maxillitis is the operation of choice because:

1. It removes a dangerous focus of infection.
2. It preserves the function of one of the most important cranial nerves.
3. It safeguards the eye against ulceration of the cornea, as a cornea that is anesthetic is particularly liable to injury even with the operation on the posterior sensory root which leaves the sympathetic fibers of the ganglion intact.

In the treatment of tic douloureux, we are dealing with a persistent condition, and therefore I present these cases merely as a preliminary report as enough time has not elapsed to judge as to the permanency of results. I trust that in the future observations of others may be added as to the relationship of chronic maxillitis to tic douloureux.

2. SPINAL CORD.

Payr, E. CONSTITUTIONAL PATHOLOGY OF THE SPINE. [Arch. f. klinische Chir., March 26, 1920.]

This lengthy article deals with various types of weak backs. The muscular and ligamentous asthenias are discussed in detail. Pains and fatigue in the back and legs, sacral pains and other complaints of typical asthenia are prominent. The children should be encouraged to exercise and play games that will strengthen the spine, not weaken it by giving them a brace or corset. Psychotherapy is essential to encourage them and make them forget and outgrow their tendency to masochistic enjoyment of their weakness; not prosthesis to confirm them in their neurasthenia. When there is actual malformation of vertebræ the sensitive organ must be spared during the more painful periods, but general strengthening measures should be kept up and the choice of a vocation supervised.

Monar, C. DANGERS OF LUMBAR PUNCTURE. [Allg. Zschr. f. Psych., Vol. LXXVI.]

Except in brain tumors the operation is without danger. On the other hand, least often in paretics, more frequently in other most varied conditions or in normal persons certain always temporary symptoms appear as a result, that is severe headache and vomiting, both lasting as long as eight days. A horizontal position is to be recommended during the puncture and should be maintained for at least 24 hours afterward. The patient should also be carried from the examination table to his bed. Quincke, strange to say, does not consider brain tumors as contraindications in spite of the cases of death observed when they are present. Others, however, surely are right in rejecting lumbar puncture in all processes that reduce space in the cranial cavity or the spinal canal.

Namack, C. SIGNIFICANCE OF YELLOW SPINAL FLUID. [Am. Jl. Med. Sci., April 1920. B. M. J.]

Yellow spinal fluid was found in a series of ninety-six cases. In six, Froin's syndrome occurred—namely immediate coagulation of the fluid on standing, yellow coloration (xanthochromia) and a marked increase in cells and albumin; two were cases of meningeal hemorrhage, two of tuberculous meningitis, and two of poliomyelitis. Froin's true syndrome represents the extreme development of a process in which yellow coloration is the earliest stage. Forty were cases of tuberculous meningitis, thirteen poliomyelitis, one meningeal hemorrhage, three cerebral hemorrhage, one cord tumor, one cerebrospinal meningitis, one pachymeningitis. The clinical observation that the presence of yellow fluid in a case with meningeal symptoms strongly suggests the probable diagnosis of tuberculous meningitis or poliomyelitis is supported.

Cooper, N. A. INJECTIONS OF CEREBROSPINAL FLUID IN CEREBRO-SPINAL FEVER. [N. Y. Med. J., December 4, 1920.]

In this clinical study the case of a boy, aged fourteen, suffering from a severe form of cerebrospinal fever is reported. He recovered from the illness following injection of his own cerebrospinal fluid. April 7 he was first observed. Treatment was nil. A lumbar puncture was made May 12 and 10 c.c. of fluid was withdrawn. It was sterile. Of this fluid 0.75 c.c. was injected subcutaneously, and it was repeated every day till the seventeenth. Gradually, the temperature fell and meningeal symptoms abated with every injection until May 18, when the temperature dropped to 97° F. The boy recovered slowly and left the hospital July 26 perfectly well.

Hall, G. W., and Callender, R. J. ARSENIC IN SPINAL FLUID. [Arch. of Neur. and Psychiatry, June 1920.]

According to Hall and others arsphenamin given intravenously in 0.6 gm. doses may be detected in the spinal fluid in from 25 to 35 per cent of the cases. Irritation of the meninges by the injection of auto-serum into the spinal canal does not increase this percentage.

Wallgren, A. XANTHOCHROMIA IN CEREBROSPINAL FLUID. [Acta Med. Scandinavica, May 6, 1920. J. A. M. A.]

Wallgren regards the hemorrhagic tendency of epidemic meningitis as one explanation of the xanthochromia observed in the cerebrospinal fluid. Or the xanthochromia may come from stagnation of the spinal fluid from obstruction of communication with the cranial subarachnoid space. In 103 cases of epidemic meningitis at the Upsala hospital in the last five years the mortality after the first twenty-four hours was 19.6 per cent, but it was only 14.8 per cent among the 74 without xanthochromia while it was 39 per cent among those with xanthochromia. The total mortality in these groups was respectively 28.3 per cent, 25, and 42.1 per cent, testifying to the gravity of the cases in which xanthochromia is observed. It forms part of what he calls the syndrome of Froin, that is, the spinal fluid is frankly yellow, with abundance of albumin, coagulates *en masse*, and contains numerous mononuclears. It not only throws light on the prognosis, but warns of the necessity for intraventricular injection of the antiserum if the condition is not improving under spinal injections. More attention should be paid to serotherapy by the vein in these cases, as epidemic meningitis is a general septicemic condition, not confined to the meninges. The details of six cases are described to sustain these conclusions and two pages of bibliography are appended. The article is in French.

BOOK REVIEWS

Ballance, Charles A. A GLIMPSE INTO THE HISTORY OF THE SURGERY OF THE BRAIN. [Macmillan and Co., London.]

Sir Charles Ballance here presents in his Thomas Vicary lecture a fascinating historical sketch of the development of cranial surgery from the remotest times to the present. It is a delightful and scholarly production.

Bleuler, E. DAS AUTISTISCH-UNDISZIPLINIERTE DENKEN IN DER MEDIZIN UND SEINE UEBERWINDUNG. Dritte Auflage. [Julius Springer, Berlin.]

The author tells us that this work was really conceived during his student days when he was often irritated by much that was taught him by his professors which was not only useless but was positively harmful.

The present reviewer recalls with much seriocomic affect a recurrent migraine which overcame him and made his life miserable at eleven o'clock in the morning nearly every time he was compelled to listen to a surgical lecture which always contained the concept of "healthy laudable pus." His entire orientation towards surgery was distorted by the nonsense expounded by this worthy and dear old man. Fortunately a wise stoicism prevented him from falling into the hands of his "eye strain" teacher or he would have become a "bespectacled" hypochondriac, or had his eye muscles cut, his teeth taken out, his tonsils enucleated, or other autistically reasoned out therapeutic procedures would have deprived him of most of his organs.

History repeats itself, and Bleuler's rather caustic criticism of autistic and undisciplined thinking as it is still widely diffused in medicine comes as a reminder that the teachings about "healthy laudable pus," "eye strain" and hundreds of other superstitions of the past have their analogues to-day.

His is a type of criticism that differs from the paranoid lucubrations of a de Krief and his ilk, for he honestly seeks to show how the affective life of the individual tends to make him believe in things which are false. Bleuler shows how our *wish* to have a thing so, is often sufficient to make it appear to us as reality, whereas it is largely phantasy; a product of our desires which undisciplined thinking makes it impossible to correct.

He thus consistently and earnestly sets forth a large series of these faulty thinking methods which still prevent medicine from being as fruitful a social enterprise as it otherwise could be. Could regular

medicine rid itself of the thinking crudities which flourish in the all too prevalent pseudomedical cults it would have an authority and following of unparalleled preëminence. As it is since it frequently is little better in its thinking than that of these cults it often fails to command the respect to which it really is entitled.

Should the principles here outlined be made the sound acquisition of medical students of the present day, we are convinced that a great advance in medical thinking would be furthered. We can conceive it to be a large gain to medicine if such a book could be translated into many tongues and its contents be made a part of an entrance requirement to the study of medicine. We might ironically suggest that the professors also be required to read it.

Bayliss, William M. THE VASO-MOTOR SYSTEM. [Longmans, Green & Co., New York and London.]

This welcome addition to Starling's excellent series of monographs on Physiology by Bayliss discusses the various factors which produce changes in the diameter of the blood vessels. The results of nerve action as well as those due to chemical agencies are detailed. The author sets himself the limited task of showing how blood pressure is modified by changes in the caliber of the blood vessels and how the blood supply of individual organs is regulated in correspondence with their needs in different degrees of activity.

The anatomy and nerve supply of the blood vessels is hastily sketched. Sensorimotor efferent supplies are indicated. Sensory nerves *to* blood vessels are stated, with receptor organs pictured. Are we captious in saying afferent impulses go *from* these receptors, rather than *to* (p. 5)? The function of the receptors in the smaller arteries is somewhat problematical at present. Peripheral resistance is discussed chiefly in physical terms. Arteriole tone also. "Blood vessels do not entirely relax when *deprived* of their nerve supply" (p. 12). Can a blood vessel be *deprived* of its nerve supply? Do the many so-called classical physiological experiments ever *deprive* a blood vessel of its receptors, or of its intravascular wall neuro-mechanisms? Do not nearly all of these experiments deal with large synthetic conglomerates rather than with the smaller isolated reactions of limited regional activities? Thus when Bayliss speaks of the rhythmic activities of blood vessels "apart from the activities of the nervous system" and speaks of these efficient stimuli as being "distension, warmth, chemical agents," do they act independently of nervous structures which are contained within the walls of the blood vessels themselves? Or does he only see a "nervous system" that must go up to the cord, or to the midbrain or to the cortex? Do Krogh's (Rouget's) contractile cells act independently of a neuro-mechanism? Being pigmented (chromatophores[?]) what are their analogies to other pigmented sympathetic (adrenalogenic) cells? Bayliss touches upon this problem (Chapters V, VI) but does not seem to orient his reasoning from the peripheral stimulus standpoint although the younger Gaskell's interesting work on the phylogeny of

adrenaline producing substances offers much material for thought from this point of view. The efferent sympathetic pathways are always emphasized, but rarely the afferent. Was Langley really wrong in denying afferent sympathetic pathways? If all the complex reactions were studied from the standpoint of afferent stimuli arising within the structures themselves as well as from the more recognized standpoint of impulses reaching them through efferent pathways would many of the contradictions here noted be resolved? The reviewer thinks so. Is inhibition a phenomenon of two reins held by a central rider, pulling now positive, now negative, or is it really a balance between afferent statements of response to internal stimuli and efferent experiential factors of more synthetic and general value to larger values as correlated in cord, midbrain or cortical centrals?

Hoffmann, Hermann. VERERBUNG UND SEELENLEBEN. EINFÜHRUNG IN DIE PSYCHIATRISCHE KONSTITUTIONS- UND VERERBUNGSLEHRE. [Julius Springer, Berlin.]

A prodigious amount of research upon the many complicated issues of heredity and constitution as applied to the medical field have been issued in the past ten years. In spite of this richness of material the carefully worked applications to neuropsychiatry still remain scattered throughout many important researches but with few exceptions have not been brought within the limited compass of a general review of the entire series of problems concerning heredity and constitution as applied more specially to psychiatry.

This book attempts such a general review. In it the author has carefully presented the material in a very thorough and yet concise manner; *i.e.*, as concisely as such complicated matters can be generalized. Hereditary Fundamentals, such as the Mendelian laws, the reasons for them, sex heredity, crossed heredity, complicated crossings, new character acquisitions, etc., are well discussed in his first chapter. The Applications to Human Conditions are taken up in the second. The Psychical Constitution is dealt with in chapter three. Constitution and Constellation, and Temperament, and Valency and Variety are here discussed with special emphasis given to Cyclothymic, Schizothymic, and Epileptoid Constitutions. Chapter IV deals with "Nervous" degenerative features as they are conditioned by embryonic, somatic, or hereditary destructive factors. The Results of Hereditary Studies are offered in the largest chapter of the book wherein are gathered data concerning the heredity of talent and genius, cyclothymic heredity, schizothymic heredity. Intermediary cyclo-schizothymic polymorps, paranoid psychotic trends, compulsive states, psychopathic states, sex perversions, epilepsies and feeble-minded states are all presented from the hereditary point of view. The final chapter deals with the practical goals of these hereditary researches. An excellent bibliography and name and subject index close this splendid monograph of over 250 pages.

Nowhere have we seen so thorough and praiseworthy an effort to encompass so complicated a group of phenomena.

Baudouin, Charles. STUDIES IN PSYCHOANALYSIS. [Dodd, Mead and Company, New York.]

Although the reviewer was not overimpressed by this author's suggestion and autosuggestion we welcome this later study since it seems to show a definite evolution in his grasp upon psychopathology. He has become more and more concrete and handles psychical mechanisms with much more precision.

We can sympathize with his eclecticism. Morons abound apparently as plentifully in Switzerland as here, and the "suggestive" appeal and the "autosuggestive," "ce passe" clap-trap may be a practical necessity over the entire world where sufficient intelligence is lacking to use the more carefully developed psychoanalytic principles.

This work therefore can be recommended as one of the more serious contributions to the psychoanalytic technic. It is highly commendable for its charming style, possibly due in part to the splendid rendering by Eden and Cedar Paul to whom we are indebted for a number of masterly translations. The case histories, which make up two-thirds of the book, are excellent.

In one respect we are convinced that Baudouin still indulges in much autistic thinking. His frequent use by the concept "normal" is evidence of this. This concept, as a projection of narcissistic absolutism, is still firmly anchored through academic teachers and we regret to see it being used as a plea to bolster up the author's estimation of the differences in "theories" as he terms them. In these days, we maintain, that it is the paranoid's special attribute to know what is "normal."

We also emphatically object to the use of the concept "sub-conscious" as employed by Baudouin, and are totally at a loss to understand what the translators mean when they prattle about "Freudian dreams." We presume they mean by this dreams with obvious erotic manifest content. As a definite hemorrhage from the bladder would result in bloody urine, while a smaller kidney hemorrhage would reveal the presence of blood by the microscope alone, so in the language of the introduction a dream with obvious erotic significance would be a "Freudian dream," whereas one that was not analyzed as to its latent erotic sources, as notably Rivèrs' dreams in his "Conflict and Dreams" would be a non-Freudian dream. Such piffle makes one wonder if the translators really know what they are talking about. Because by proper analytical methods Brown finds the presence of gold in an ore and Smith is unable to do so, one should thereby distinguish the same product as Brownian or Smithian ore.

What right has Baudouin to claim (p. 7) that functional activities are sequels to structural activities? Are not organs structuralized experiences? Without experience, *i.e.*, function, there would be no structure; function is constantly moulding and leading structure, rather than the reverse. They are reciprocating mechanisms, in which "time" as a functional unit, provides the constantly new stimulus

which makes and modifies structure and permits it to accumulate a "mneme."

If evolution as an hypothesis is preferable to preformation, then function must be considered a determiner for structure; if the reverse, then structure determines function. A billion years ago, the earth is inferred to have been but a molten mass of 80 chemical elements still whirling in space. It is true that man now has structurally utilized 28 of these 80 structures; and whereas the chemical combinations of phosphorus and sulphur and others do make up the structural arm that guides this pen they do not determine the function of the writing. Baudouin never really gets into the functional point of view. He cannot seem to envisage it without the fear that he will do injustice to descriptive formulations. Others who do are in danger of "mere theorizing." The hypothetical nature of all scientific formulations is hard for him to uphold without the fear of sliding into something not approved of by pedants.

Maxwell, S. S. LABYRINTH AND EQUILIBRIUM. [J. B. Lippincott Company, Philadelphia and London.]

This, the latest of the Monographs on Experimental Biology, edited by Loeb, Morgan and Osterhout, aims to present in short and compact form an objective study of the reactions of vertebrate animals to gravity and to show the mechanisms by means of which these reactions are made possible. Fishes were used mainly in this study, selachians offering the best opportunity because of their size.

The author has confined his study chiefly to the mechanisms as seen in vertebrates although the subject of the evolution of the labyrinth from invertebrate forms could not be neglected entirely.

His discussion of the gradual evolution of the otolith organ is not as satisfactory as one would like it, but in view of the difficulties in bridging the gap between invertebrate and vertebrate evolution the position taken is conservative at least. We are inclined to believe that Winkler's treatment of the problem is more stimulating for the actual worker with pathological problems.

This is but a small digression, however, since Maxwell has not really set himself the task of presenting the entire problem of the labyrinth and equilibration. What he has done is to give us an extremely readable and accurate account of the chief situations.

Gehring, John George. THE HOPE OF THE VARIANT. [Charles Scribner's Sons, New York. \$2.00.]

It is with a sigh of relief that the reviewer lays down this really most fascinating book, a relief born of the conviction that among the tons of semipopular literature on nervous ills, here is one finally that shows real intelligence. Here is a book conceived as a true naturalist should conceive his problems. Problems in "variations," not as the pedantic academic regards them as "normal" and "abnormal"; these atrocious hypothetical standards of inquisitorial origin. Gehring is not satisfied to count and weigh, he values.

Human beings vary infinitely. Human understanding that can take hold of those variations which lead to discomfort and distress and change them is true understanding and leads to true healing. It is no wonder that Gehring has had his great success. He is a real psychiatrist and not an overspecialized "reverse cripple."

His book is a joy to read. It is not overtechnical; it certainly is not dryly pedagogic, it may even be said to lack an appreciation of the profundity of certain problems of adjustment and great variation, but it can be read with profit by all.

Lewandowsky, Max. PRAKTISCHE NEUROLOGIE FÜR AERZTE. Vierte Auflage von R. Hirschfeld. [Julius Springer, Berlin.]

Lewandowsky's untimely death—he died of typhus during the Great War—left his great Handbook and his smaller works half revised and reëdited. Hirschfeld, whose excellent grasp of the subject of neurology is reflected in his masterly editing of the Zentralblatt f. d. ges. Neur. u. Psych., has revised the text of the "Practical Neurology" in a more thoroughgoing manner than he did for the third edition in 1919. He has brought it closer in line with the great advances made chiefly as a result of that war. It stands to-day as a complete general discussion of modern neurology in short, concise and philosophical form. Its neurological foundations are better conceived than its psychiatric formulations. In this respect the author lags behind the comprehension of the dynamic psychology and rattles the bugaboo of pansexuality to bolster up the resistances to the insight into human personality that Freud's researches have made possible.

Schneider, Kurt. DIE PSYCHOPATHISCHEN PERSÖNLICHKEITEN. [Franz Deuticke, Leipzig und Wien.]

The Handbuch der Psychiatrie, edited by Aschaffenburg, has been revived after a pause due to the war. Two new fascicles have been received.

This one by Professor Schneider of Cologne, about 100 pages in size, deals in thoroughly orthodox Kraepelinian fashion with the concept of "Psychopathic Personalities." The importance of understanding the outstanding problems connected with the behavior of this general personality type admits of no doubt. Like the poor they are always with us. Their behavior is in need of supervision through many agencies. To properly deal with them requires the wisdom of the serpent and the patience of Job. Some of this wisdom is here presented to us. We regret that it is cast somewhat in the old orthodox intellectualistic molds. There is much talk of normal and abnormal, and almost nothing of the development of personality from the natural history or dynamic point of view. Thus the book is excellent for legal definition purposes, but it imparts no real understanding of the problems involved. Ferenczi's important contributions are not mentioned in the index even, nor are any other ideas than those of the old school German, permitted to enter. With

these limitations it is an excellent presentation of this development in psychopathology.

Stekel, W. CONDITIONS OF NERVOUS ANXIETY AND THEIR TREATMENT. Authorized Translation by Rosalie Gabler. [Dodd, Mead and Co., New York.]

When it is realized that it has taken 15 years for Stekel's original work on *Nervöse Angstzustände* to be put into English dress one can appreciate its essential vitality on the one hand and the slowness of dispersion of knowledge concerning the newer movements in psychopathology. In this latter respect at least two facts are pertinent. The great war held up all forms of international exchange of intellectual progress and Stekel's own volumes were so large that publishing customs prevented their translation and issuance. Fortunately by breaking up his volumes into smaller units this author's valuable writings are now being made available.

The present work on anxiety states was one of the first clinical offerings of the newer psychoanalytic school. It is rich in illustrative material and shows Stekel at his best. The lessons it contains should be more widely diffused. The neuropsychiatrist knows much of this material; to the internist it is largely a closed book and he goes on treating psychical conditions with organic expressions by various superstitious survivals. He is the practical man of whom Lord Beaconsfield speaks, "as one who continues to practice the errors of his forefathers."

We can hope for a wide reading of this valuable book; no matter in what smaller matters of style or of argument we might prefer to have had it otherwise, the chief point is that it strikes at the heart of countless situations to-day which if the practitioner of this country had been alive to their real significance and not been led by the nose by autistic thinking professors of internal medicine, the countless fads and cults and religious treating of human disorders would never have been possible.

Hart, Bernard. THE PSYCHOLOGY OF INSANITY.. [Cambridge University Press, Cambridge.]

We are no more pleased with the title of this book than we were ten years ago when it appeared, but it was evidently a good "selling" title, for this is the third edition and the eighth reprinting of this edition originally appearing in 1916.

It is a great pity with such a good selling title that the author has not really brought it in line with consistent concepts which have been crystallized since it was first printed. It was almost the first work in England to launch itself on the waters of the Freudian libido stream. Even if this was done only halfheartedly and with certain apologetic fears, it should be time for Hart to make this little manual even more useful by revising it, pruning out the barbarous word "insanity" and making it, not a perpetuator of this medieval mind attitude towards a living problem, but an agency for enlightenment. So long

as intelligent psychiaters talk of the psychology of "insanity," there is little hope of getting out of our medico-legal morass of misunderstanding. English lunacy practice may not suffer from this difficulty but in the United States "insanity" as a medical concept is an impossibility.

Scripture, E. W. STUTTERING, LISPING AND CORRECTION OF THE SPEECH OF THE DEAF. Second Edition. [The Macmillan Company, New York.]

Stuttering is a mental disturbance. It is a neurosis; it is not an organic disorder involving the speech mechanism. Those organic tics which may result from encephalitis and which may superficially resemble stuttering can readily be eliminated if one studies the speech mechanisms carefully.

Scripture has done more for the accurate study of vocalization than any other American investigator. He is well qualified to speak of the disorders of speech and this new edition is a welcome testimony of the value of its teachings.

Miller, H. Crichton. THE NEW PSYCHOLOGY AND THE PARENT. [Thomas Seltzer, New York.]

This is a popular exposé of a diluted analytic psychology in words of one syllable, as it were. It is a very good one of its kind and can be recommended to inquiring parents.

Pear, T. H. REMEMBERING AND FORGETTING. [E. P. Dutton and Company, New York. \$2.50.]

In England, as in the United States, the great war brought into its vortex a great number of workers from fields entirely outside of those usually involved. Among these psychologists have shown in their written records that they learned a lot about human beings which they had not before realized, having been more or less isolated within academic confines, mural as well as intellectual. Professor Pear of Manchester has already contributed a study with Dr. Elliot Smith on the war neuroses as a result of his participation in medical work and he here presents an interesting book on the psychopathology of everyday life, chiefly dealing with Remembering and Forgetting. Chapters on "Dreams" with too much reliance on Rivers, who really was an amateur in psychoanalysis and other psychological topics are dealt with.

There is a certain lack of coherence in these studies which, as the author tells us, were written at various times between 1913 and 1921, but on the whole the book is much more readable and instructive than many we have met for some time.

OBITUARY

PAUL WATERMAN, M.D.

Dr. Paul Waterman, of Hartford, Conn., died July 31, 1923, of acute dilatation of the heart, after a strenuous career. He was only forty-six; born at Westfield, Mass., December 17, 1877. He obtained the degree of B.A. at Williams, and M.D. at Cornell. After general hospital experience at Bellevue he was assistant resident alienist there for several years and then studied a year in Vienna, becoming while there president of the American Medical Association of Vienna. After his return to this country he practiced neurology and psychiatry at Hartford, Conn., and held many positions of honor and activity in hospitals and medical societies, and in the militia of Connecticut. In 1915 he was graduated from the Army Field Service School for medical officers at Fort Leavenworth, and for four months in 1916 was with the federalized Connecticut troops in Arizona on the Mexican border. He joined the regular federal service in 1917 and became assistant division surgeon, 26th Division, under General Edwards. In France he was made medical liaison officer of the division at the headquarters of the 11th Corps d'Armée and held that post from February 11 to March 15, 1918. Then, after serving as assistant to the chief surgeon of the Yankee division, he became commanding officer of the 101st Sanitary Train, which played an important part in much of the heavy work of the division. On October 1, 1918, he was assigned division surgeon, 4th Division, and on the day of the Armistice commissioned lieutenant-colonel. On March 5, 1919, he was made colonel, and honorably discharged from the U. S. Army August 26, 1919, after twenty-one months in France.

His localized service in France comprised the Chemin des Dames Sector, La Rène Sector (with three actions), Pas Fini Sector (near Chateau-Thierry), Champagne-Marne Defensive, Aisne-Marne Offensive, Rupt Sector (near St. Mihiel), St. Mihiel Offensive, Troyon Sector, Meuse-Argonne Offensive, and with the Army of Occupation. After his return to the United States Colonel Waterman continued to live a strenuous life and was loaded with honors, medical, civil, and military, in the State. He was a member of 23

societies, clubs, and associations, and wrote several papers chiefly on intracranial injuries and on sanitation. He maintained his membership and interest in the New York Neurological Society for the 20 years he was a member, although unfortunately his distant residence prevented his attendance.

CHAS. E. ATWOOD

F. MARTIUS

Prof. F. Martius, for many years director of the University Clinic in Rostock, died in October at the age of seventy-three. He was appointed staff physician in Gerhardt's clinic and a university instructor in 1887, became assistant professor and director of the medical policlinic in Rostock in 1891, and in 1892, after the death of Thierfelder, director of the medical clinic. Martius is best known for his interpretation of modern constitutional pathology. In his autobiography, published in the beginning of this year, he says, "In the theory of pathognostic hereditary transmission, I think I may rightly claim to have been a pathfinder and pioneer worker." He first expressed his views in 1889 in an address "Krankheitsursachen und Krankheitsanlagen," and he has supported and developed them since in many articles and addresses. His paper on the subject before the Congress of Internal Medicine, in 1905, was especially noteworthy. His other researches have been in the fields of diseases of the heart, stomach and nervous system. His so-called marking method, proposed in 1888, constitutes an original method of examination in the diagnosis of heart disease. His researches on the quantitative determination of the gastric contents have been embodied in a monograph, "Die Magensäure des Menschen," published in collaboration with Lüttke in 1892. The wide extent of his literary activity is evidenced in his autobiography, which lists sixty-eight articles and treatises. Martius possessed a high type of intellect and was distinguished for his critical abilities. His pleasing manner procured him many friends, but his frank defense of his views also brought him many vigorous opponents. At the end of his career he had the satisfaction of knowing that many of his new views had been accepted.—(Berlin Letter, J. A. M. A.)

NOTES AND NEWS

SICARD AND GUILLAIN

Two new professors have just been appointed to the Paris Faculty—J. A. Sicard to the chair of medical pathology, and G. Guillain to that of neurology. Both are young men. Is it not rather remarkable that as we grow older we see the highest posts occupied by teachers who seem to be always growing younger?

Professor Sicard will continue to be the driving force he has always been. The welcome given him by the students at his recent inaugural address is a guarantee of this. He was working with Widal when the latter established the principle of the serum diagnostic test for typhoid fever, and for a quarter of a century he has devoted himself to the study of the cerebrospinal fluid. If our professors were required to choose a coat of arms, those of Sicard should bear a syringe and a long needle! To him we owe the method of the blocking of the nerves by the injection of alcohol. Only the other day he used the transcerebrofrontal method to introduce anti-meningococcal serum into the region of the base of the brain itself. His is the honor of introducing the spinal injection of lipiodol, which has made it possible to determine the exact site of a tumor of the cord with the aid of X-rays. Helped by the information thus obtained the surgeon can boldly cut straight down upon the lesion.* *Audaces fortuna juvat.*

Upon Professor Guillain has fallen a mantle difficult to wear. The fame of the neurological school at the Salpêtrière has drawn to the grey walls of that monumental hospital pilgrims from all corners of the world. Hard is the succession to a line of teachers which stretches from Charcot to Pierre Marie. Guillain, however, is a born neurologist, and many publications on anatomy and physiology have come from him. He has tracked and unravelled a series of reflexes by which the science of symptomatology has been enriched. Long, too, have been his labors in the pathology of the nervous system, and fruitful their results. In his inaugural address on December 20, he combated the popular opinion that neurology is an art rather of diagnosis than of treatment. He showed that neurology had a

* Paris Correspondent B. M. J.

positive side, and then went on to give his audience a picture of those unchartered territories which are awaiting pioneers. As a first consideration he asked, What is the nature of the nervous impulse itself? He ended by quoting Parker of the Johns Hopkins University, who has just proclaimed that Europe, bled white, is incapable of keeping her place in the domain of science, and must hand on the torch to America, which has the strength, the enthusiasm, and the dollars! "We do not yet feel ready to have it snatched from our hands," said he, and his words will find an echo in British hearts. Let me add that Professor Guillaïn is very well acquainted with the English-speaking scientific world, and a convinced advocate of international coöperation. He is one of the very few of our Paris teachers who is able to lecture as well in English as in French. He is well worth watching.

Dresden: Following the retirement of Professor Dr. Ganser (our account of his death being "grossly exaggerated") as Director of the Dresdener State Hospital, Dr. Reiss from Tübingen has been appointed as chief of the First Division Receptor's Wards, Dr. G. Flatau as director of the Second Division (Chronic Psychoses) and Dr. Schob of the Third Division, Chronic Nervous and other organic diseases.

Frankfort a/Main: Professor Dr. Kleist, director of the psychiatric clinic at Frankfort, has received a call to fill the chair of Professor Bumke at Leipzig.

Dr. Huchzermeyer, the director of the combined Bethel-Bielefeld Epileptic Colonies, has just died at the age of seventy-two.

The Journal OF Nervous and Mental Disease

An American Journal of Neuropsychiatry, Founded in 1874

ORIGINAL ARTICLES

NEUROSYPHILIS AS AN ETIOLOGICAL FACTOR IN THE PARKINSONIAN SYNDROME *

BY HUGO MELLA, M.D., AND S. E. KATZ

BOSTON, MASS.

The etiology of the Parkinsonian syndrome, excepting that due to epidemic encephalitis, is obscure. Syphilis has rarely been considered an etiological factor, but that a relationship may exist between these two conditions has lately been pointed out by various observers. Jeanselme, (1) gives a clinical description of a case of neurosyphilis with symptoms of encephalitis lethargica which later developed the Parkinsonian syndrome. He points out the difficulties in making the diagnosis of encephalitis lethargica in the presence of syphilis as one is too ready to ascribe the symptoms to neurosyphilis. Guillain (2) describes two cases with positive Wassermann tests in blood and spinal fluid that simulated encephalitis lethargica; both improved under antiluetic treatment. Archard, (3) and Renaud, (4) discuss mesencephalic syphilis simulating epidemic encephalitis. Schaffer (5) describes a case of paralysis agitans in a syphilitic; his spinal fluid abnormalities responded readily to antiluetic treatment, but the treatment had no effect on the clinical manifestations; tremors, rigidity, micrographia, gait, and speech remaining unchanged. Schaffer believes that there is no relationship between the syphilitic infection and the clinical manifestation of paralysis agitans. Many observers have reported positive Wassermann reactions during the acute stage of encephalitis lethargica which later, with the subsidence of the symptoms have become negative. None of these cases gave

* From the Department of Neuropathology, Harvard Medical School.

other evidence of syphilis, and none of these observers reported pathological findings.

On the Neurological Service of the Massachusetts General Hospital two cases appeared during the month of June, 1923, presenting symptoms of paralysis agitans. They gave vague histories that might be interpreted as symptoms of encephalitis lethargica three or four years previously; and both had definite syphilitic histories. One of these two had been treated in this hospital ten years ago for neurosyphilis. Each of these patients gave a negative response to the blood and spinal fluid Wassermann test. The diagnosis of postencephalitic paralysis agitans met with greater favor than the diagnosis of paralysis agitans with a syphilitic etiology.

Only one histological description of syphilitic lesion of the basal ganglia has been found in the literature: Loeper (6) reports the pathological changes in the caudate nucleus of a syphilitic patient who had no symptoms of paralysis agitans. He describes a large number of obliterated blood vessels with small hemorrhages in the parenchyma some of which were organized, indicating a chronic process.

Inquiry at various pathological laboratories has shown that the neuropathological investigation of syphilis has usually been limited to a study of the cortex, medulla and spinal cord, neglecting the basal ganglia. It therefore seems worth while to give a clinical report with pathological findings of a case of active neurosyphilis presenting the Parkinsonian syndrome.

CASE

J. R.—a white man of forty-six years—was admitted on June 30, 1922, to the Boston City Hospital with a diagnosis of "paralysis agitans." His family history was essentially negative. He gave a history of chancre ten years ago. Three years ago the patient began to have slight pains in both legs and arms. At that time he also noticed that his legs commenced to twitch. He went to the Massachusetts General Hospital where a positive blood Wassermann was found and there received fifteen intravenous injections of salvarsan. His tremors continued and gradually increased during the week before entry. He was in the hospital for a week and on the fifth day developed pneumonia, from which he died.

General Examination: A middle aged, well developed, but emaciated man with a mask-like face. Heart, lungs and abdomen negative, except before death (when pneumonia developed).

Neurological Examination: Tremor involving face, arms and legs; some rapid coarse tremor of the jaws; fine jerky tremors of the tongue; both arms and left leg showed coarse, rapid tremors (pill rolling type). Tremor diminishes slightly with intention.

Eyes: Left pupil irregular and smaller than right; both pupils fixed to light and reacted to accommodation. Left choroiditis.

Reflexes: Arm, abdominal and cremasteric present and active. Knee jerks and ankle jerks absent. No Babinski or ankle clonus. Incontinence.

Mental: Confusion, depression, delusion, visual hallucinations and suicidal ideas.

Laboratory Findings: Blood Wassermann, July 1, 1922, doubtful; July 3, 1922, positive. Spinal fluid pressure 4 mm. of mercury. Cells 52. Wassermann negative. No other tests made.

Autopsy Findings: General: Bilateral bronchopneumonia with edema of lungs and fibrous pleurisy; sclerosis of aorta.

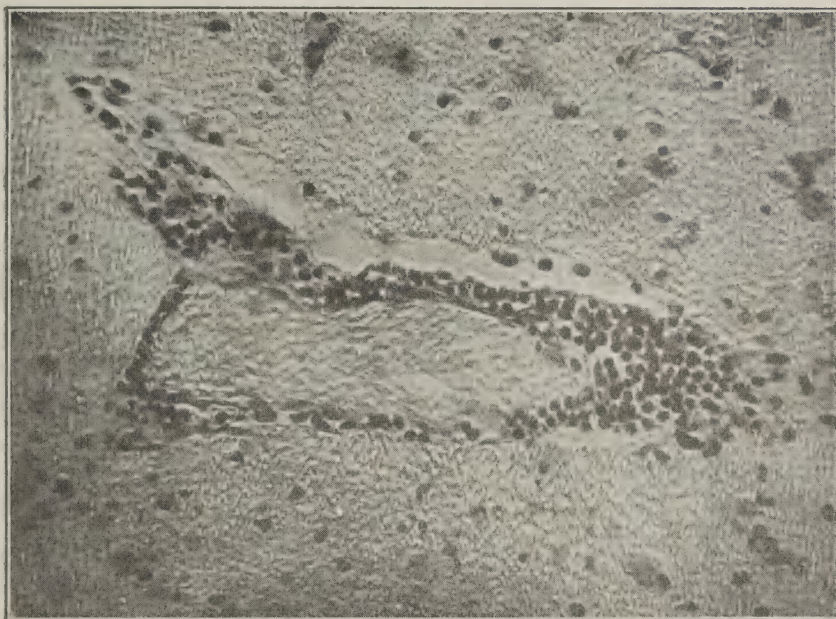


FIG. 1. Section of putamen illustrating the degree of perivascular exudate.

Central Nervous System: In the gross, chronic leptomeningitis, atrophy of frontal convolutions, and slight granulations in fourth ventricle were observed. Brain weight, 1,485 grams. Dura adherent to pia in two places in frontal poles. A small amount of fluid found in subdural space. Blood vessels showed no gross abnormality. One small plaque was seen in the basilar artery. The arachnoid and pia were opaque and edematous over convex surfaces of the cerebral hemispheres. Anteriorly the two hemispheres were bound together by adhesions of the pia, especially at the base of the brain. Many miliary foci of thickening stand out on the pia. The convolutions in the frontal regions are narrow and the sulci wide; while in the posterior portions both appear normal. Basal ganglia and lateral

ventricles showed no gross lesion on section. Floor of fourth ventricle showed a slightly granular surface. Cerebellum showed no gross abnormality.

A careful microscopical examination of the basal ganglia was made. In the putamen and pallidum there are numerous new capillaries. The nuclei of the endothelial cells are large and extend into the lumen of the capillaries. The old blood vessels show very marked thickening of the endothelial lining as well as that of the outer layer.

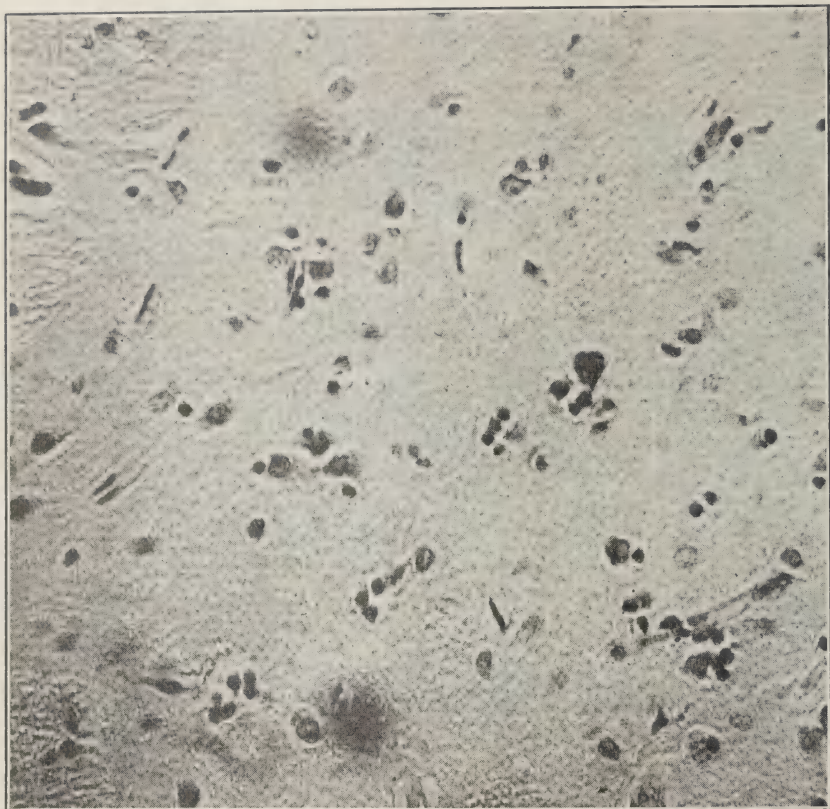


FIG. 2. Section of pallidum showing rod cells. In many instances it is difficult to differentiate between the rod cells of glial origin and these degenerated nerve cells. In a single high power field one is able to trace the degenerative process, finding cells with very little change, cells with moderate changes, and finally cells which resemble rod cells.

There is marked perivascular infiltration. The cellular element is mostly of the round cell type mixed with many plasma cells.

The cellular elements show a definite decrease of the pallidal type of cell (Golgi Type 1). The cells are few in number and those remaining show marked degenerative changes. The changes are intranuclear as well as extranuclear. The nuclei of some are pyknotic,

others show chromatolysis, the nuclei being swollen and pale with the nucleoli deeply stained, in marked contrast to the rest of the nucleus. The cytoplasm is shrunken, staining faintly and the Nissl bodies have mostly disappeared. The general outlines of the cells are very much distorted. Some of them have undergone such a marked degree of degeneration that they closely resemble rod cells.

There is marked neuronophagia, from one to four glia cells surrounding each nerve cell. Many of these glia cells have fine vacuoles and others take the stain with a yellowish tint. Evidently these are signs of active phagocytosis. The glia cells are mostly of the small

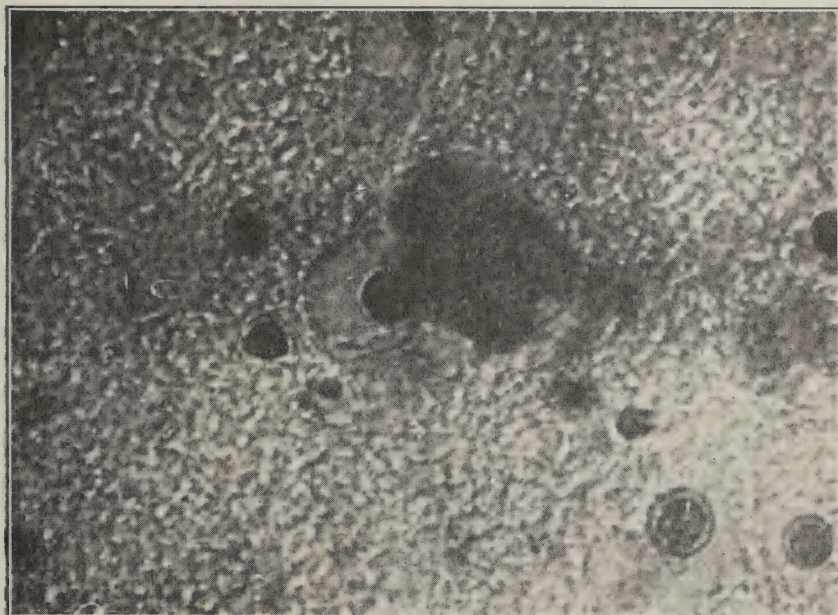


FIG. 3. Section from pallidum illustrating neuronophagia. The small nerve cells (Golgi Type 2) although involved in the pathological process, are less affected than the larger cells. Their number is somewhat decreased. The general cytoplasm and nuclear changes are the same as those described for the large cells.

type described by Buzzard and Greenfield,(7) with many rod cells among them. Hematoxylin eosin stain brings out the marked glial proliferation. There is an apparent increase in the connective tissue elements; in practically every high power field there are cells that cannot be classed as either glia cells or degenerated nerve cells. These are cells with only a little cytoplasm that takes on a pink stain with Cressyl violet; they have an oval nucleus that stains purple. These cells may be derived from the connective tissue elements of the numerous new blood vessels.

Not infrequently one encounters an ameboid glia cell, as described by Buzzard and Greenfield,(7) which has a pale, large

nucleus with fine chromatin material around the periphery. The cytoplasm is small in amount, takes a faint stain, and in it appear many vacuoles. The cell outline is irregular, having numerous processes.

In sections of the cortex and pons the blood vessels show a perivascular infiltration mostly of the round cell type, with few plasma cells. The walls of the blood vessels are very much thickened and show proliferation of cells in the adventitia. There is a marked increase in glia cells and the nerve cells show various degrees of degeneration with a considerable number in a good state of preservation.

DISCUSSION

In this case we have two types of lesions microscopically. First, a perivascular infiltration of round cells and rod cells in the pallidum; and secondly, nerve cell degeneration. The first is indicative of neurosyphilis. This is borne out by the gross findings of chronic leptomeningitis, atrophy of frontal convolutions and granulations in the fourth ventricle which are pathognomonic of neurosyphilis. At our present state of knowledge we are not able to state that these two types of pathological changes belong to one disease entity. It is probable that the neuron changes are secondary to the vascular changes. On the other hand, it may be that the neuron change is a primary one like that found in presenile paralysis agitans and that we have two pathological processes going on at the same time without any definite association. To determine this, the basal ganglia of a series of syphilitic brains should be studied to determine the frequency of pallidal cell degeneration in syphilitic patients who do not show paralysis agitans symptoms.

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PERSONAL OBSERVATIONS AND CRITIQUE OF THE
DIAGNOSTIC SIGNIFICANCE OF THE
LABYRINTHINE REACTIONS *

BY SAMUEL J. KOPETZKY, M.D., F.A.C.S.

AND

RALPH ALMOUR, M.D.

NEW YORK CITY

The definite standardization of the methods of examining the labyrinthine mechanism brought the otologist to a realization of two factors: first, that he was conducting his research in a virgin field which promised a rich harvest; and secondly, that he had heretofore been devoting his attention to a mechanism which is an integral part of the nervous system and ignoring its central connections while he concentrated only on its peripheral end mechanisms. As he realized this, what was to be expected happened. The labyrinth tests were applied to each and every variety of intracranial lesion. In the course of time a series of findings were evolved which the otologist considered pathognomonic for certain given conditions. In many instances the proof of the correctness of his labyrinthine syndromes was so striking and brilliant, because the diagnoses were made in the absence of the more usual signs, that the otologist became exceedingly enthusiastic and pronounced these tests the *prime factor* in the diagnosis of brain lesions. In doing this, the otologist encroached upon the fields of neurology to such an extent that it was impossible to view him in any light other than that of a diagnostician-in-chief for intracranial conditions.

Having assumed this position, he was naturally called upon to diagnose brain lesions of all kinds. In a great many instances he failed so miserably in eliciting his pathological responses in cases of intracranial tumors which were later proven either by operation or autopsy findings, that his tests rapidly fell into disrepute with the greater number of neurologists and, to a certain degree, with otologists also.

Just as the Wassermann reaction, which at first was considered

* From the Oto-Laryngological Dept., Beth Israel Hospital, N. Y. Presented before the Section on Otology, New York Academy of Medicine, December 14, 1923.

to absolutely decide the presence or absence of syphilis, has gradually assumed its place as a link in a chain of symptoms, its absence not necessarily excluding a luetic condition, so have the labyrinth tests now become, instead of the deciding factor in brain lesions, a diagnostic aid in localizing and recognizing them.

Much has been written, but much more remains unknown, concerning the fundamental principles involved in the production of the labyrinthine reactions. It is not the purpose of this paper to enumerate them; neither are we here concerned with the spontaneous phenomena which are supposedly due to a disturbance of the vestibular mechanism. In this paper we will deal only with the pathological responses elicited by stimulation of the labyrinth *by rotation and by douching with cold water*.

NYSTAGMUS

The following, at this time, may be considered as being established and generally accepted:

1. A destruction of any portion of the vestibular nerve, either the central nuclei in the medulla, the nerve trunk or the end organ, will result in an absence of after-nystagmus on stimulation by means of the caloric method.

2. The after-nystagmus which results from the stimulation of the normal labyrinth by turning with the head forward thirty degrees and ninety degrees, is of two varieties, horizontal and rotary. The motion of the eyeball in horizontal nystagmus takes place in the horizontal plane, the slow component being produced by the contraction of either the internal or external rectus muscle. The rotary motion of the eyeball occurs in the frontal plane, the vestibular component being due to the contraction of the superior and inferior oblique muscles. Therefore, if, upon stimulation of the labyrinth by rotation in the horizontal and frontal planes, a nystagmus other than a rotary or a horizontal one is obtained, it must be due to a lesion in or about the brain stem which affects the ocular muscle nuclei directly, since, from clinical evidence, only the sixth nerve nucleus and those portions of the oculomotor nucleus which innervate the internal rectus and the superior and inferior oblique muscles receive impulses from the labyrinth activated by rotation in these planes.

3. Since stimulation of the horizontal canals normally produces a horizontal nystagmus and excitation of the vertical canals with the head forward ninety degrees elicits a rotary nystagmus, the finding of a perversion of the after-nystagmus points to a lesion of that

portion of the vestibulo-ocular tract that is located between the vestibular and ocular muscle nuclei in the brain stem.

There have been observations made concerning the nystagmus reaction, both clinical and physiological, which until recently were accepted dogmatically. They have been the basis for many of the so-called labyrinth syndromes. It is the axiomatic character of these reactions of the labyrinth to stimuli that first led us to doubt some of our own findings, to believe that we had erred in our technique, and to formulate diagnoses that were impossible and improbable when considering the case as a whole. The postulates to which we refer are as follows:

1. *It has been held that when the caloric stimulus fails to produce a reaction when the head is tilted backward, while causing a reaction in the upright position, or vice versa, it is evidence of a central interference with the vestibulo-ocular tract.*

In a careful survey of the literature, we find that the usual routine method of examining the labyrinthine function is by rotation and by douching the ears with cold water. The rotation test is almost always limited to turning the patient with the head thirty degrees forward, thereby testing the horizontal semicircular canals. The caloric test is usually performed with the water at sixty-eight degrees, the head at first thirty degrees forward and then tilted sixty degrees backward. The assumption is that the vertical canals are tested individually by douching with the head placed thirty degrees forward, and that, by tilting the head sixty degrees backward, the horizontal canals are individually tested.

By examining in this way the estimation of the function of the vertical canals by rotation is totally neglected. We are in the habit of testing the canals by rotation in both the horizontal and the frontal planes, in this way checking our caloric findings, and have as a result obtained interesting observations. The following cases may be cited as elucidating the point at issue:

Case I. M. B., age twenty-one, suffering from congenital lues. During the past five years she has become progressively deaf, so that at present hearing is *totally lost in the left ear*, while it is still present to a slight degree in the right ear.

The spontaneous vestibular and neurological reactions were normal.

The rotation tests elicited *no reactions whatsoever from either the horizontal or the vertical canals*. The caloric test, while showing no response from either ear when the head was placed thirty degrees forward, showed, when the head was placed back sixty degrees, a *rotary nystagmus to the left* following stimulation of the *right* ear,

and a slight but *definite horizontal nystagmus to the right* following the stimulation of the *left* ear.

Case II. J. L., age twenty-one, one and one-half years before examination received an injury to the right side of his head, following which he had a typical attack of diffuse purulent labyrinthitis which gradually abated, leaving him *totally deaf* in the right ear after both the internal and the middle ear suppuration had abated.

The spontaneous vestibular and neurological reactions were all normal.

The rotation tests were as follows:

To Test Horizontal Semicircular Canals

Turning to right:

Nystagmus: Thirty-five seconds to left, horizontal, rapid.
(Checked.)

Vertigo: Eleven seconds to left.

Pastpointing: Normal; with both hands to right.

Turning to left:

Nystagmus: Twenty seconds to right, horizontal. (Checked.)

Vertigo: Nine seconds to right.

Pastpointing: Normal; with both hands to left.

To Test Vertical Semicircular Canals

Turning to right:

Nystagmus: Twenty seconds to left, rotary.

Vertigo: Fourteen seconds to left.

Pastpointing: Normal; with both hands to right.

Falling: To right.

Turning to left:

Nystagmus: Eight seconds to right, rotary. (Checked.)

Vertigo: Sixteen seconds to right.

Pastpointing: Normal; with both hands to left.

Falling: To left.

The caloric test showed that while there were no reactions when the right ear was douched with the head thirty degrees forward, a *normal horizontal nystagmus to the left* appeared when the head was tilted backward sixty degrees. The left ear showed normal responses.

Summarizing, we have in the first case no responses from any of the canals by rotary stimulation, while the caloric test produces a reaction when the head is tilted backward sixty degrees. In Case II we have definite evidence of a loss of function of the right semicircular canals, by rotation, as shown by the diminution of the duration of the nystagmus on turning *away* from the diseased side as compared with turning *toward* it; and the caloric test, while produc-

ing no reaction when the head was thirty degrees forward, gave a definite nystagmus when the head was placed sixty degrees backward.

These cases present findings which are so contradictory that one is at a loss to estimate their value. In the first case, the rotation tests showed that the semicircular canals were not functioning. Yet, on caloric stimulation, it would seem that impulses from both horizontal canals reached the ocular muscle nuclei. The second case definitely shows on rotation that the right semicircular canals are dead; yet by the cold caloric one obtains a reaction, when the head is tilted backward, which would lead us to believe that the horizontal canal functions and that our rotation test was at fault. The latter was checked, with the same result at each test.

Undoubtedly many observers have at times found discrepancies between the rotation and caloric tests but were at a loss to explain them, many attributing the findings to a faultiness either of their technique or of their observations. Barany observed these conditions but offered no explanation for them, admitting that his theory of the caloric reaction did not account for such findings nor properly explain them. Cases very similar to the ones we have observed have been reported by Beck, Ruttin, Neumann, Urbantschitsch and Borreis,(1) who advanced various theories for their explanation.

The cases reported by Borreis are very analogous to our own in that the reactions following rotation were absent while those following caloric stimulation were present. He considers that the two reactions do not issue from the same part of the labyrinth. He proved experimentally (2) that, in pigeons, after the removal of the semicircular canals, no reaction whatever could be elicited by turning; such reactions had disappeared forever. These same pigeons, however, produced normal caloric nystagmus when the external auditory canal was syringed. To him this proved that the caloric stimulus arose from some portion of the vestibule, probably in the otolith organs. This showed further that the cristæ ampullæ were not necessary for the production of the caloric nystagmus.

That the semicircular canals function primarily for the perception of *angular* movements is sufficiently evident when one considers their anatomical arrangement, which includes the three planes of space. We know that by rotating an individual in a certain position a nystagmus is produced that cannot be altered by any change in the position of the head. We further know that the normal person will show movements of the eye in an opposite direction to the position of the head, first described accurately by Barany as *Gegenrollung* (or counter-rotation) (3). We also know that the caloric nystagmus

is dependent for its character on the position of the head in space. Therefore, since after caloric stimulation the character of the resultant nystagmus is dependent upon the position of the head in space and not upon the perception of any movement of the body in space, since there is definite proof that caloric nystagmus can be elicited in the absence of the canals, since we know that the "counter-rotation" of the eyes disappears when the otolith membrane is destroyed (4), as has been demonstrated by Magnus and de Kleijn (5), we conclude from all the premises that the caloric nystagmus is the result of the stimulation of the otolith organs in the vestibule.

Finally, the natures of the two stimuli are different. The stimulus by rotation is one of motion, while the caloric stimulus is one of temperature. Motion causes stimulation of the labyrinth in such a way that the end organ registers movement about an axis, with the resultant characteristic compensatory phenomenon of endeavoring to keep the visual field the same as before rotation. This is definitely dynamic in character. The caloric stimulus, on the other hand, is the result of temperature changes in the internal ear; and the end organ registers, not movement about an axis, but changes in the internal ear due to a change in the status of the endolymph, most probably its density, analogous to changes in the density in any fluid that is subjected to sudden change of temperature. While this may cause a disturbance in the canals, Maxwell (6) feels that a still larger disturbance is produced in the larger mass of endolymph in the vestibule, with consequent tension upon the membranous labyrinth and the otolith organs.

One must not underestimate the importance of the observations of Borreis. They bring us an entirely new aspect of the labyrinth tests. Heretofore we have adhered to Barany's conception of the production of the caloric reactions as originating in the cristæ ampullæ and have interpreted our findings accordingly. There have been developed tract systems for the various canals on the basis of Barany's interpretation of the caloric reactions, which led us to diagnose intracranial lesions much as an electrician discovers a short-circuit or a break in a wire. Barany's theories seem to hold true when all the reactions are proper, but they fail when we are confronted with what Borreis terms the "paradoxical vestibular reactions."

In view of the findings, namely, that the caloric reaction can be produced in the absence of the semicircular canals, that the rotation tests may be absent while the caloric are present, that the nature of

the two stimuli are different in that one produces reactions by movement of the body about an axis while the other causes a reaction by a change in temperature, it must be acceded that these two reactions, one of which we will term the "angular reactions" and the other the "caloric reactions," emanate from different parts of the labyrinth. Therefore we must alter not only our established interpretation of these reactions, but we must also consider as not definitely proved that system of tracts which have been developed for the individual canals. Since it is inconceivable that the caloric reaction is produced only by the semicircular canals, we cannot interpret the absence of a caloric reaction as being due to an impairment of the canals or of the supposed central connections of the canals. While it is true that clinically we have observed that certain caloric reactions will be absent in intracranial lesions, we cannot account for their absence as signifying a disturbance of the *tracts leading from the canals*, as we have heretofore been doing. Perhaps the central fibers in the brain stem are the same for the canals and for the otolith organs; but the observations of Borreis tend to show otherwise. In the future, we will estimate the significance of the tests by analyzing the "*angular nystagmus*" and the "*caloric nystagmus*" separately, giving to each its particular worth in the diagnosis of brain lesions. At present, with this end in view, we simply record our observations.

2. The second postulate supposes that a unilateral destruction of the labyrinth will result in a shortening of the duration of the after-nystagmus on turning away from the diseased side as compared with turning toward it, the ratio being approximately two to one.

We desire to report two cases in which there was a unilateral destruction of the labyrinth with findings which do not accord with this.

Case III. M. L., age thirty-nine, has been totally deaf in the left ear for approximately thirty-five years. The caloric test showed definitely that the left labyrinth was not functioning, while the right one produced normal reactions. The rotation tests do not show what we would ordinarily expect, *i.e.*, a reduction in the time of after-nystagmus on turning to the right as compared with turning to the left, but *equal reaction time* for the turning in *both* directions.

The findings in detail were:

Turning to the right:

Horizontals: Seventeen seconds to left.

Verticals: Eighteen seconds to left.

Turning to the left:

Horizontals: Eighteen seconds to right.

Verticals: Nineteen seconds to right.

Case IV. P. G., age forty, sustained a fracture of the skull three months before examination. The caloric test produced no response from the right labyrinth after douching for five minutes. This was checked a number of times, always with the same result. Hearing in the right ear was limited to the perception of very loud shouting, and that very indistinctly. He was not totally deaf. The rotation tests were as follows:

Turning to the right:

Horizontals: Eight seconds to left.

Verticals: Fourteen seconds to left.

Turning to the left:

Horizontals: Six seconds to right.

Verticals: Fifteen seconds to right.

This brings out an interesting point. Here in the first case we have a man who has had a dead labyrinth practically all his life. From repeated clinical observations we know that a destruction of the labyrinth will produce a diminution in the duration of after-nystagmus on turning away from the diseased side with the head at thirty degrees forward, because the maximum stimulation arises from the side opposite to the direction in which the patient is turned. In this case, however, taking the findings at their face value, we have an absolute contradiction to what is usually accepted as definite. Taking into consideration, though, the period of time during which the labyrinth has been inactive, we conclude that while an early examination of a destroyed vestibular mechanism will reveal the characteristic findings, an examination performed some time later will show a readjustment and compensation on the part of the healthy labyrinth analogous to any other part of the human anatomy that has two organs performing the same function. The second case illustrates the same point, the only exception being in the duration of the inactivity of the end organ. Ruttin (24) and Eagleton (7) have mentioned this; the latter has termed it "establishment of compensation."

3. The third postulate, based on Ewald's experiments on pigeons, *assumes that a current AWAY from the ampulla in the vertical canals is capable of producing TWICE as strong a reaction as a current toward it.* In other words, the results obtained are found to be opposite to those which obtain when the horizontal canals are stimulated. Clinically we find that the postulate holds true for reactions obtained by turning when testing the horizontal canals, but we do not find it holding true when the verticals are similarly tested.

When the head is brought forward ninety degrees, the superior and posterior canals are placed as nearly as possible in the horizontal

position. In this position, the ampulla of the superior canal is external to the nonampullated end, while the ampulla of the posterior canal is internal to the nonampullated end. It has been demonstrated and accepted that when the vertical canals are tested in this manner conjointly, the endolymph movement is in the same direction in reference to the ampulla in both the posterior and superior canals on one side. That is to say, if the current is toward the ampulla in the superior canal, it is toward the ampulla in the posterior canal of the same side.

In reference to the horizontal canals, we arrive at the conclusion that the current *toward* the ampulla produces the stronger reaction as follows:

1. *Anatomy*: The ampulla of the horizontal canals are external to their nonampullated ends.

2. *Endolymph Movement*: We know that the movement of the endolymph continues in the same direction as the turning, after the sudden stopping of rotation.

3. In examining a case where there is a unilateral destruction of the labyrinth, turning toward the diseased side, thereby causing the endolymph to move toward the ampulla on the healthy side, produces a reaction twice as strong as turning away from it.

By applying the same principles to the study of the vertical canals during rotation, we find the following:

1. *Anatomy*: The ampulla of the vertical canals are situated so that the ampulla of the superior canal is external, while the ampulla of the posterior canal is internal, to the common nonampullated end.

2. *Endolymph Movement*: We have seen that if the endolymph movement is toward the ampulla in the superior canal, it is also toward the ampulla in the posterior canal on the same side.

3. On turning a patient who has a unilateral destruction of the labyrinth, we find that on turning toward the diseased side the reaction elicited is approximately twice as strong as that obtained when turning away from it. The following cases will illustrate the point we desire to make:

Case V. A. R., age forty-two, totally deaf in the *left* ear.

The caloric test elicited no responses from the left labyrinth after douching with cold water for four and one-half minutes.

To Test Horizontal Semicircular Canals

Turning to right:

Nystagmus: Ten seconds to left, horizontal.

Vertigo: Fifteen seconds to left.

Pastpointing: Normal; with both hands to right.

Turning to left:

Nystagmus: Eighteen seconds to right, horizontal.

Vertigo: Thirteen seconds to right.

Pastpointing: Normal; with both hands to left.

To Test Vertical Semicircular Canals

Turning to right:

Nystagmus: Eight seconds to left, rotary.

Vertigo: Twenty seconds to left.

Pastpointing: Normal; with both hands to right.

Falling: To right.

Turning to left:

Nystagmus: Fourteen seconds to right, rotary.

Vertigo: Twenty-two seconds to right.

Pastpointing: Normal; with both hands to left.

Falling: To left.

Case VI. M. S., age twenty-five, a case of cerebellar abscess reported by Drs. Kopetzky and Schwartz (25), in whom, following purulent labyrinthitis, all hearing was lost in the right ear.

The caloric test elicited no responses from the right labyrinth.

To Test Horizontal Semicircular Canals

Turning to right:

Nystagmus: Twenty-four seconds to left.

Vertigo: Nine seconds to left.

Pastpointing: Normal; with both hands to right.

Turning to left:

Nystagmus: Sixteen seconds to right, horizontal.

Vertigo: Ten seconds to right.

Pastpointing: Off with the right hand, to right. Normal with left hand, to left.

To Test Vertical Semicircular Canals

Turning to right:

Nystagmus: Thirty seconds to left.

Vertigo: Eight seconds to left.

Falling: Slight objective falling to right.

Turning to left:

Nystagmus: Eleven seconds to right.

Vertigo: Seven seconds to right.

Falling: Objective falling to left.

Case II, reported elsewhere in this paper, will also serve to elucidate this point.

In all these cases, we notice that the turning toward the diseased side produced a reaction greater than turning away from it, not only in the horizontal canal, but in the vertical canals also. In the first case, turning toward the left, that is, toward the destroyed labyrinth, caused the current to move toward the left after the rotation had been stopped. Moving toward the left, the endolymph impulse is therefore toward the ampulla in the right superior canal, and consequently toward the ampulla in the posterior canal. The duration of the nystagmus obtained by turning toward the left was almost twice as great as that obtained by turning toward the right. The other two cases show identical findings.

From this we conclude that the endolymph impulse in the vertical canals is capable of producing the strongest reaction when it is in the direction *toward* the ampulla. This would be much more logical, since it shows the function of the cristæ of the vertical canals to be the same in character as is ascribed to them in the horizontal canals.

THE SHORTENING OF THE DURATION OF AFTER-NYSTAGMUS

It has been demonstrated by MacKenzie (8), and also by Jones (9), that the stimulation of the normal horizontal canals produces an after-nystagmus of approximately twenty-four seconds duration. Our observations have been in accord with this, rather than with the results reported by Barany. We have also found that stimulation of the vertical canals by rotation yields an after-nystagmus of approximately fifteen seconds duration. Any appreciable variation from these figures must be accounted for. It is true that some perfectly normal individuals will show an after-nystagmus that is either very short or very prolonged. This we must consider as peculiar to their normal physiological status.

In the examination of cases of undoubted increased intracranial pressure, Eagleton (10) has found that, where the increase in pressure is located in the posterior fossa, the duration of the nystagmus obtained after turning is diminished by almost 50 per cent of the total obtainable nystagmus. This he attributes to the impairment of a center located in the cerebellum which controls the duration of rotation nystagmus. We have found that in all cases where we obtain an appreciable decrease in the duration of the after-nystagmus elicited by rotation, we also notice an increase in the time required to produce a reaction by douching with cold water; in other words, it takes longer for a given caloric stimulus to cause a reaction. For example, if turning to the right (thereby eliciting the greatest response from the left) results in a marked diminution of the period

of after-nystagmus, *it will take a longer period of time than the approximately normal to produce a nystagmus from the left labyrinth by means of the cold caloric method.* A citation of two cases will serve to illustrate the point. As we are concerned only with the nystagmus, we will omit the other findings.

Case VII. J. B. had sustained an injury to the back of his head, since when he complained of constant occipital headache. On examination he presented a moderate ataxia of the upper extremities.

Turning him to the left, thereby eliciting the maximum response from the right canals, resulted in a horizontal nystagmus of eighteen seconds' duration, as compared with thirty seconds obtained when turning him to the right. It took one minute and twenty-five seconds to produce a nystagmus by douching the right ear with cold water, as compared with forty-five seconds for the left.

Case VIII. I. W., a man in whom a definite diagnosis of syphilis was made.

On turning him to the left, the duration of the nystagmus was twenty-three seconds shorter than on turning him to the right. The caloric test required two minutes to excite a nystagmus from the right ear, as compared with fifty seconds for the left ear.

Here we are confronted by an observation which requires explanation both as to its physiological and its mechanical origin. It would seem, from our findings, that a direct relationship exists between the duration of the after-nystagmus from rotation and the time required to produce a nystagmus with the cold caloric stimulation. We are disinclined to accept the theory of Eagleton that a center exists in the cerebellum governing this function.

Were Eagleton's contention true—*i.e.*, that there exists in the cerebellum a center that controls the duration of the rotation nystagmus—coupled with our observations that where the duration of the after-nystagmus is shortened there will be found also a diminished excitability of the labyrinth to the cold caloric, it would at first appear as if this phenomenon were the result of the impairment of a cerebellar center. That the cerebellum has no effect on either the production or the character of the nystagmus other than that of maintaining the tonus and synergia of the ocular muscles, has been definitely shown by De Kleijn and Magnus (11), who demonstrated that nystagmus following rotation occurred normally in the absence of the cerebellum. Then again, if this phenomenon is the result of the impairment of the function of a center in the cerebellum, this center must have a two-fold action, one which controls the capacity of the ocular muscle nuclei for the reception of stimuli, and one which

would control the amount of stimulation necessary to produce a reaction.

There is a remarkable specialization of brain tissue cells, each so-called center acting for guidance of one, and only one, function. Because of this truism, one can point out definitely the portion of brain tissue involved when a certain function is lost. Again, the caloric and rotation nystagmus are essentially different, as we have seen, in that the one concerns what we call "angular movement" and the other has to do with position in space. In view of these physiological factors, and in view of the relationship which our observations lead us to think exists between the duration of the after-nystagmus from rotation and the time required to produce a nystagmus by the caloric stimulus, we believe that this phenomenon of "shortened-nystagmus-time—increased-caloric-time" is due to an interference with the retro-labyrinthine fibers to the ocular muscle-nuclei, causing a diminution in the *force* of the transmitted impulses, rather than to an impairment of a center in the cerebellum to which we would have to attribute a dual function to make its activity logical and comprehensible. This theory would not affect the findings of Eagleton in cases of increased intracranial pressure, but would offer a more reasonable explanation for them; namely, that they are caused by the pressure acting on the brain stem, resulting in the impairment of the transmission of the impulses, rather than that the increased intracranial pressure acts on a given center located in the cerebellum.

ABSENCE OF THE QUICK COMPONENT

Fisher and Jones (12) incline toward the theory that the center for the quick component is located in the cerebral hemispheres and that it is within the realm of consciousness. MacKenzie (13) also adheres to this view, considering the movement as purely volitional. Barany (14), on the other hand, emphatically states that the rapid component is not due to the cerebral cortex, citing a case of bilateral hemiplegia wherein the quick component was demonstrable. He believes that the return movement is entirely of subcortical origin. In support of this, Bartels (15), viewing the finding of Tozer and Sherrington (16) of sensory fibers in the nerves which supply the ocular muscles, believes that the impulses which produce the return phase of nystagmus pass to the ocular muscle nuclei through these fibers, thus completing a reflex movement.

An interesting case, which tends to support the cortical control of the quick component, recently came under our observation.

Case IX. A patient of Dr. Altman, who, on operation, proved to have an endothelioma of the dura pressing upon both frontal lobes and invading the left frontal lobe.

The caloric tests showed that while all other reactions were normal, the quick component was absent, the tests resulting only in a conjugate deviation toward the side stimulated.

Whether the tumor, by virtue of its destruction of tissue or by the pressure exerted upon the brain, caused the center for the quick component to lose its function, is not at present clear. We present this case merely to put on record that an absence of the quick component was found in a case of frontal lobe tumor.

VERTIGO

The production of vertigo by stimulating the labyrinth is not very satisfactory as an aid in the diagnosis of brain lesions. As yet, we can interpret only its absence, and that very vaguely. We know that vertigo should and must be present following stimulation of the labyrinth. When vertigo is absent, then something is wrong; but from it alone one cannot deduce even in the remotest way what or where that something is. We have made the following observations:

1. The after-vertigo obtained by stimulation of the vertical canals by turning is of appreciably shorter duration than that elicited from the horizontal canals by turning.

2. After destruction of one of the labyrinths, the duration of after-vertigo obtained by rotary stimulation is appreciably diminished; but what is true for after-nystagmus, namely, a reduction in the duration of after-nystagmus on turning *away* from the dead labyrinth as compared with turning *towards* it, does not apply to vertigo. Turning in one direction will produce the same, or nearly the same, duration of after-vertigo as on turning in the opposite direction. *Vide* Cases II, V and VI.

3. In caloric stimulation we have observed that the patient complains of a sense of vertigo *after* the nystagmus has been present for some time, and that the vertigo is of much longer duration than that obtained by rotary stimulation.

4. People with a definite nervous trend will normally show a markedly prolonged after-vertigo. Professional dancers, on the other hand, have a marked tolerance for turning-stimulation; and do not exhibit vertigo readily upon rotation.

These observations are not, however, of any definite diagnostic value. Vertigo must be studied through the reactions that it produces, namely, pastpointing and falling, for we have at present no definite means of determining pathology by means of variations in the character and time of vertigo.

PASTPOINTING

Analysis of Pastpointing: Of all the reactions looked for after labyrinthine stimulation, that of pastpointing is probably the most confusing and is regarded by many observers in an unfavorable light because of the inability to accurately diagnose, by means of it, the exact location of intracranial lesions. It is in the interpretation of the results of induced pastpointing that one can find the real basis for the controversy between the otologist and the neurologist. We are not as yet firmly established in our knowledge of the manner in which the physiological factors of pastpointing are influenced by labyrinthine excitation. We have not here, as in nystagmus, a definite reflex arc, with a definite reflex which is the direct result of stimulation of the labyrinth, and which is almost foolproof in its pathology. On the contrary, we have here a very complex act, which is dependent, not upon the labyrinthine impulses, but upon the vertigo that is produced by stimulation coupled with a conscious, coördinated motor volition.

It is possible, however, on the basis of what we already know of the functions of the various parts of the brain, to arrive at certain conclusions which are logically and physiologically true.

The mechanism of induced pastpointing has four phases, each of which must be normal in its entirety for the reaction to be performed properly.

1. Stimulation of the vestibular mechanism results in a disturbance of the individual's conception of his position in space.

2. When we now ask the patient to raise the extremity to be tested, we call into action his motor power.

3. The muscle groups used act in a synergic manner, the coördinating impulses for which arise in the cerebellum.

4. Consciousness, imagining that external objects are moving away, causes the patient to point towards the place where he thinks the fixed object is now located.

A disturbance of *any one* of these phases will result in a faulty pastpointing. A destruction of the labyrinth with its resultant absence of vertigo or any other condition wherein vertigo is not elicited by stimulation of the end organ will cause pastpointing to be absent. A disease of the motor apparatus, be it central, peripheral or in the muscle itself, will result in either absent or faulty pastpointing after stimulation.

When a patient, in the presence of normal vertigo and normal motive power, fails to pastpoint in the normal direction, we immediately attribute it to a dysfunction of the cerebellum, reasoning that

since there is accurate proof that there are certain centers in the cerebellum which control the inward and outward movements of the extremities, the seat of the trouble must be in the vicinity of these centers. We do know that the cerebellum is concerned with the control of the synergic action of all the body musculature, and we further know that it exerts its action directly on the muscles without the intervention of consciousness, by means of the cerebello-spinal tracts which end in the ventral horn cells. These tracts are three in number, the vestibulo-spinal, thalamo-spinal and rubro-spinal tracts (17). Governed by the cerebellum, through the efferent fibers which pass through the inferior peduncles to Dieter's nucleus, and by fibers which pass through the superior cerebellar peduncles to the thalamus and red nucleus, wherein lie the nuclear cells of these tracts, the latter act normally in such a way as to assure a continuously harmonious equilibratory status.

We have, then, three separate and distinct pathways for the transmission of cerebellar impulses to the efferent spinal nerve routes. With such a wide territory, where a disturbance of any portion will produce a loss of coördination, how can we with purely clinical data at our command attempt to accuse the peduncles and the cerebellum for every variation from the normal pastpointing. The afferent vestibular channels and the cerebellum proper may be free from pathology, but a lesion along the efferent spinal tracts will cause a disturbance of the synergic action of the muscles. For instance, we know that a thalamic lesion, such as is found in the syndrome of Dejerine-Roussy (18), will produce some degree of hemiataxia, showing a faulty synergic control.

Since the work of Bolk (19) on the function of the cerebellum in animals, confirmed by Van Rijnberk (20) and the more recent investigations of Barany (21) in the human, leading to his identification of definite areas associated with special muscle actions, the otologist has concentrated on the cerebellum and its afferent tracts to the exclusion of the efferent cerebellar channels. However, from a histological standpoint, this localization cannot be as distinct as that in the cerebral cortex.

Each Purkinje cell is connected with the afferent cerebellar tracts by means of the climbing fibers. According to Tilney (22), these individual cells are concerned with single synergic units. An intercommunication is also established between groups of Purkinje cells by means of the basket fibers. Still larger grouping of these cells is made possible through their granule cell connections. In this way the cerebellum, while capable of producing simple synergia through

the single synergic units, functions primarily in regulating the "patterns of movements" rather than the parts employed. While "there may be areas which regulate adduction, abduction, flexion, extension as such; these are to be regarded as parts of more comprehensive regions controlling movements of the extremities in all planes produced by the action of synergic units." (29)

OBSERVATIONS

1. We have found that, in cases of proven syphilis of the central nervous system, a definite interference with the normal pastpointing was invariably elicited upon rotary stimulation despite the absence of other cerebellar signs of either asynergia or dysmetria. This is mentioned here merely as an observation in four cases, further investigation being necessary to bring evidence sufficiently conclusive.

2. We have also noted that differences in the pastpointing may be found upon repeated examinations in the same individual.

3. If there is a disturbance of pastpointing on stimulating the horizontal canals by rotation, one usually elicits the same disturbance when the vertical canals are stimulated.

GENERAL CONCLUSIONS

1. To the otologist, the labyrinth tests are of extreme value in determining the amount of function present in the end organ. They enable him to diagnose with a great degree of certainty the various diseases of the labyrinth *per se* and act as a guide in determining the time and mode of operation on the end organ in suppurative processes.

2. In neurology these tests have their place, but their value is more or less limited because of our present lack of definite histological evidence to verify and clarify our clinical observations and substantiate our theories.

Lesions below the tentorium, when they involve the vestibular pathways, will produce disturbances in the labyrinth reactions which are definite enough to be diagnostic. These cases, however, with the exception of primary eighth nerve tumors, will give other signs which are of diagnostic value. Cerebellar lesions, when they are large enough, will evince themselves by pressure on the brain stem, causing interference with the transmission of impulses along the vestibular tracts. Off-pastpointing following stimulation, in the absence of other neurological signs, is of little value in diagnosing these conditions, as there are usually present more marked disturbances of coördination, such as adiadochokinesis, a positive Romberg

or Babinski's dysmetria reactions, which would label the tumor as cerebellar in origin much more easily than does the pastpointing test. We feel, therefore, that where we get a pastpointing in the wrong direction following stimulation, and where the spontaneous pastpointing is normal, we are not justified in our present state of knowledge in endeavoring to render an opinion as to the situation or cause of the lesion.

In supratentorial tumors, the tests are only of negative value. While it has been found that rapid invasion of the frontal and temporal lobes may produce disturbances in the pointing reactions, the effects are too transient to enable us to consider them as diagnostic evidence of disease in these parts.

We feel, however, that every case of intracranial lesion, wherever possible, should be subjected to oft repeated labyrinth tests for the purpose of recording gradual changes in the reactions. In those cases where we are fortunate enough to verify the presence of tumor, whether by operation or autopsy, an exceedingly careful gross and microscopic examination of the portions of the brain tissue involved by the growth should always be made with a view of determining a histological explanation for the disturbed labyrinth responses. Too often has the otologist failed to establish or verify a diagnosis of brain tumor where the tests should have given lucid evidence of the disease. Very rarely could this be attributed to faulty technique or careless observations. We are at present groping about in a maze of clinical observations, trying to determine the cause from the effect, without having even tried to explain pathologically how and when, if at all, the cause produces the effect. We have probably been treating this problem in an erroneous manner. Until we can find a definite pathological explanation for the abnormal variations of the labyrinth reactions, until we can with certainty attribute to various portions of the central nervous system the control of these reactions, until that time will there be constant quibbling, constant controversy and an everlasting dispute as to the value of these tests.

SUMMARY OF OBSERVATIONS

1. We can corroborate the findings of Borreis in what he calls "paradoxical reactions," and further believe with him that the caloric stimulus and the rotary stimulus produce their reactions by affecting different parts of the labyrinth. We feel that the caloric nystagmus and the rotary nystagmus are different from each other both as regards character and point of origin.

2. After a period of time, more or less variable, there occurs in a patient with only one functioning labyrinth a readjustment and compensation on the part of this labyrinth, analogous to the compensation which takes place in any other part of the body that has two organs performing the same function.

3. We cannot agree with Ewald's hypothesis that the current away from the ampulla in the vertical canals is capable of producing a stronger reaction than the current towards it, for our clinical observations not only fail to bear it out, but tend to show that the reverse is true. From our findings, the cristæ ampullæ in all the canals react to endolymph movement in the same manner; namely, when the current is toward them, the reaction is stronger than when the current is away from them.

4. There apparently exists a direct relationship between the duration of the after-nystagmus elicited by rotation and the time required to produce a nystagmus by the caloric method.

5. We have been able to demonstrate the absence of the quick component of nystagmus in a case of frontal lobe tumor.

6. While destruction of one labyrinth diminishes the period of after-vertigo, there is no difference to be noted in the duration of vertigo on turning away from the diseased side as compared with turning toward it. What holds true for the duration of nystagmus following the destruction of one labyrinth does not hold true for the vertigo.

7. During caloric stimulation, vertigo appears after the nystagmus has been elicited.

8. From the cases we have examined, it appears that a syphilitic involvement of the central nervous system causes faulty pastpointing after labyrinth stimulation despite the absence of spontaneous signs of asynergia or dysmetria.

9. If there is a disturbance of pastpointing elicited by stimulating the horizontal canals by rotation, one usually elicits the same disturbance when the vertical canals are stimulated.

10. We feel that, at present, no definite conclusions can be drawn from induced pastpointing where the spontaneous pointing is normal.

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EPIDEMIC ENCEPHALITIS AND SYPHILIS: DIFFERENTIAL DIAGNOSIS *

BY ALFRED GORDON

PHILADELPHIA

DIFFERENTIAL SYMPTOMS IN 18 CASES OF ENCEPHALITIS

I. Wassermann Reaction		II. Lymphocytes	III. Fever	IV. Somnolence
Blood — 14 cases		15-45 cells (5-10 later)	Present	Prolonged period
Cer.-sp. fl. + 6 cases		135-150 cells (75-90 later)	Absent	Pronounced only two weeks

V. Eye Symptoms

Cases with negative Wassermann = (a) Diplopia: late; fluctuates or disappears; (b) Ptosis: late; disappears; (c) Arg.-Rob.: disappears.

Cases with positive Wassermann = (a) Diplopia: early; persistent; (b) Ptosis: early; persistent; (c) Arg.-Rob.: persistent.

VI. Motortract

(a) K.J. (b) Achilles (c) Plantar (d) Ankle-clonus (e) Myoclonia

Cases with neg. Wass.	++	++	-(+ later)	Variable	++
Cases with pos. Wass.	++	++	+(- later)	Variable	--

VII. Sphincters

Cases with negative Wassermann	--
Cases with positive Wassermann	++

VIII. Pain

Cases neg. Wass.	++	} Lasted 10-15 days	Delirious and confusional outbreaks. Dullness.
Cases pos. Wass.	++		

IX. Mental Phenomena

X. Parkinson Syndrome

Cases neg. Wass.: (a) Hypertonia +. (b) Tremor: slight; lasts indefinitely.
Cases pos. Wass.: (a) Hypertonia +. (b) Tremor: marked; eventually disappears.

Up to quite recently a symptom-group presenting extraocular manifestations or an involvement of other cranial nerves, indicating an invasion of the pyramidal pathway, showing a slight meningeal reaction, also some alteration in the mental activity—briefly speaking, a combination of all such manifestations had been usually and almost invariably considered to be of luetic origin. Since the advent of lethargic encephalitis the situation has changed and there is a tendency at present to attribute the above mentioned phenomena among which

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there are almost always ocular symptoms, to encephalitis. A careful reading of records in the former and in present literature suggests that both tendencies were and are excessive and sometimes extreme. Nevertheless one must admit that the analogy between the two morbid conditions is sometimes very striking and the differential diagnosis becomes very difficult and delicate.

With this object in view eighteen cases were submitted to a careful and prolonged study and observation.

Let us consider in detail the various manifestations as observed in this series of cases and endeavor to determine the diagnosis of the affection from its course, duration, variability, and manner of response to treatment of the morbid phenomena.

The *Wassermann reaction* was positive in six cases and negative in twelve cases. In all the cases the blood and the cerebrospinal fluid were examined twice during the course of the disease. The positive reaction of the six cases was in four in the cerebrospinal fluid and in two in the blood and spinal fluid. In spite of the negative findings antiluetic remedies were administered and in three out of the twelve cases the symptoms disappeared completely. The symptomatology was so strikingly similar to the clinical picture of the cerebral syphilis (as formerly conceived) that in spite of persistent negative Wassermann reactions the special treatment brought no appreciative results and did not modify the course of the affection. Parallel with the Wassermann test the gold reaction was carried out in each of the eighteen cases. It was positive in the positive Wassermann cases and in two of the negative Wassermann cases. The successful antiluetic treatment in the negative Wassermann cases leads to this conclusion that either a negative Wassermann test is not absolutely pathogenic or else the mercurials and neo-arsphenamin are curative remedies even in nonluetic lesions. Exclusive reliance on a negative Wassermann cannot be considered in making a differential diagnosis between lethargic and syphilitic encephalitis.

Lymphocytosis was marked (135-150) in the six cases with positive Wassermans. It was also present in the three cases with negative Wassermans in which the antiluetic treatment gave satisfactory results. In the latter group the number of lymphocytes ranged from 15 to 45. The interesting feature from a diagnostic standpoint lies in the stability or in the instability of the lymphocytes. In the six positive cases the lymphocytosis remained high although not so marked as in the beginning (75-90). In the other cases the former 15-45 cells decreased to 5-10 per c.mm. It is evident that lymphocy-

toxis and especially its persistence in spite of the treatment, is a good and reliable diagnostic point in favor of syphilis, but not of lethargic encephalitis in which the lymphocytosis is not stable. *Fever* was present in all cases except the six with positive Wassermann, and was maintained for many weeks, although it fluctuated.

Eye symptoms: (a) Diplopia was present persistently in the six cases with the positive Wassermann reaction. It was present in all the other twelve cases but not persistently. In the former it appeared very early. In the latter later. In the former it was very obstinate and rebellious in treatment. In the latter it disappeared promptly in those ten cases in which the antiluetic remedies were without benefit, but the diplopia fluctuated (disappeared and reappeared at various intervals) in the other three cases in which the antiluetic treatment was beneficial although the Wassermann reaction was negative.

(b) Ophthalmoplegia was present in the largest majority of cases (12) but in various degrees, and affected irregularly various ocular muscles: Ptosis was present in the six positive Wassermann cases and in ten other negative cases. It appeared early in the former, but later in the latter. It persisted in the former, but gradually disappeared in the latter and more rapidly in those which were under antiluetic treatment. The third nerve was more frequently involved than the fourth or the sixth nerves.

(c) Most instructive was the observation made on the pupillary reactions. A typical Argyll-Robertson pupil (loss of light reaction and preservation of accommodation reflex) was present persistently in the six cases with the positive Wassermann reaction. It was also present for many weeks in all the other cases with this difference, that in all, except two, the typical pupil disappeared long before all the other symptoms commenced to improve. In two cases the Argyll-Robertson pupil persisted a long time after a complete recovery from all other symptoms but it eventually also disappeared.

Somnolence was present in all the cases, but not equally profound. In the six cases with the positive Wassermann it was more pronounced than in all others, but only during the first two weeks, but subsided considerably afterwards and soon disappeared. In other cases it lasted a considerably longer time and there was a marked fluctuation in its intensity at different periods of the malady.

Motor tract involvement was evident in every case of the entire group. A notable difference was observed in the stability and instability of certain manifestations. The patellar tendon reflex and

tendon Achilles reflex were exaggerated in fourteen cases at the beginning of the disease and continued so for many months. In four with negative Wassermann reactions they became normal when treated with antiluetic remedies but became again abnormal during the latter period of the affection. The plantar reflex also varied: in the cases with the positive Wassermann it was at first extensor in type but later became normal. In the other cases it was flexor at first, then doubtful, later extensor and still later normal. This variability was observed not only in the elicitation of the reflex by Babinski's method but also by other methods. In the latter case the reflex did not always run parallel with the Babinski.

Ankle-clonus showed still greater variability in the cases with the negative Wassermann. It would disappear and reappear.

Myoclonic movements were noticed in eleven cases of the group with a negative reaction. They affected the extremities and face. In four cases they persisted for months, but in all others they rapidly disappeared.

Pain existed during the first ten or fifteen days in five cases, in two of the positive Wassermann group and in the three cases in which the Wassermann was negative, but the antiluetic treatment gave satisfactory results. The pain was confined to the face and to the cervical portion of the neck. It was of a neuralgic character, very severe, and lasted but a brief period of time.

Mental phenomena consisted of a dullness and hebetude, also of delirious outbreaks. The latter were present only in the cases with a negative Wassermann reaction and only in those which had febrile attacks. Ten such cases presented a slight elevation of temperature (99.2–100.2). The fever existed only in the beginning of the disease and it was not continuous: it appeared only in paroxysms and was always accompanied by an increase of neuralgic pain in the three cases (mentioned above), also by an acute delirious state.

Sphincter disturbances existed only in the six cases with a positive Wassermann, viz., retention followed by imperative micturition. When other symptoms commenced to improve, the sphincter disorder made rapid improvement.

Parkinson's symptom-group was present in all the eighteen cases, with this difference, that in the six positive Wassermann cases it was more complete than in the cases with negative Wassermann reaction. In the former one observed complete hypertonia of the entire musculature of the body, characteristic facies with marked unilateral

tremor. In the latter one observed the hypertonia with the typical facial expression, also loss of automatic movements, but very slight tremor. The latter remained insignificant for a very long time but became pronounced later in the course of disease. In the former the Parkinsonism became greatly ameliorated and finally the tremor disappeared but a slight hypertonia remained. In the latter the tremor continued parallel with the muscular rigidity for a very long time: any improvement that occurred was simultaneous in both chief elements of the affection: in the tremor and the hypertonia.

Analysis of the symptoms and differential diagnosis. Attention is called to the positive Wassermann reaction in the spinal fluid of six cases. The latter is a powerful argument for lues. However, cases have been reported in which lethargic encephalitis developed in syphilitic individuals but superimposed on syphilis. In all such cases the encephalitis is not of a luetic character but is an independent affection. In the six individuals of our series various syphilitic manifestations made their appearance at various periods and among them the group of symptoms usually observed in encephalitis lethargica rapidly developed. It was evident that they were of luetic nature. Subsequent events proved it to be correct. This contention was corroborated by the character of the cellular findings in the spinal fluid. Not only the number of lymphocytes was quite large before the treatment was instituted, but it remained high (although somewhat decreased) throughout the course of the disease and in spite of the treatment. A difficult diagnostic situation was found in three other cases when in spite of the negative Wassermann the antiluetic remedies proved to be beneficial. Here the Wassermann test was of no special aid in the diagnosis. The cellular condition of the spinal fluid although not very high in the beginning (15-45) improved greatly and reduced the number of cells to normal. In this group of cases doubt remains as to the luetic or nonluetic nature of the encephalitis, since on one hand the Wassermann was negative and on the other the specific treatment influenced the condition favorably and reduced the number of lymphocytes to normal. What especially speaks in favor of the possible luetic character of the encephalitis is the stability of the normal number of cells once the reduction had been made. In the cases of the lethargic encephalitis, which were distinctly of nonluetic character, the cellular count was not stable; it was quite variable during the course of the affection.

In reflecting upon the eye manifestations one finds that although they are similar in cases with positive and negative Wassermanns,

nevertheless there is a fundamental difference; the latter was uniformly observed in the entire series of eighteen cases. In the positive cases diplopia, ptosis, and Argyll-Robertson phenomenon appeared early and remained persistent in spite of the treatment. In the negative cases the same symptoms while present, nevertheless appeared late, fluctuated for a short period, and eventually disappeared completely. The latter characteristic was observed even in the negative cases which were favorably influenced by the antiluetic treatment.

Particularly interesting is the Argyll-Robertson reaction. Although Sicard denies its occurrence in encephalitis lethargica, nevertheless a number of authors admit the possibility of its existence. Netter speaks very emphatically. Economo equally admits its occurrence in many cases. Nonne says that one must think of encephalitis in presence of Argyll-Robertson pupil when the Wassermann is negative in the cerebrospinal fluid. Naef observed absence of knee-jerks and the presence of Argyll-Robertson in cases of encephalitis during the epidemic of Munich. Achard's case is especially illustrative in this respect. The patient developed a general malaise, diplopia, and somnolence interrupted by delirious outbreaks. There were also a slight optic neuritis in the left eye and an Argyll-Robertson pupil so that the ophthalmologist made the diagnosis of tabes. However, the symptoms were recent, had an acute onset with some fever. The cerebrospinal fluid showed no lymphocytosis and a negative Wassermann. The Argyll-Robertson pupil disappeared during convalescence and there was no trace of optic neuritis at the end of three months. Guillain had already called attention to the existence of Argyll-Robertson's sign in nonsyphilitic lesions of the cerebral peduncle. He thus explains the phenomenon since peduncular lesions are frequent in epidemic encephalitis (*Revue Neurologique*, No. 7, 1923, p. 1040).

In the present series the Argyll-Robertson symptom was present in all the eighteen cases but it disappeared more or less rapidly in the twelve cases which presented negative Wassermans. Somnolence, although it was present in all the cases without exception, nevertheless the duration and the intensity were not identical; in the cases with negative Wassermans the symptom was of very long duration, while in the positive cases it lasted but two weeks and was very pronounced and deeper from the very beginning of the disease.

Parkinson's syndrome, like some of the previous symptoms, presented differential features. In the cases with negative Wassermans

the hypertonia appeared first in the face and gradually descended to the shoulders. It was associated with a very fine and a minimum of tremor. These two symptoms ran parallel the first two or three months and then gradually the tremor became much slighter, but the hypertonia became more pronounced and invaded the entire body. Both symptoms persisted indefinitely. Moreover, there were also respiratory disturbances, explosive laughter and considerable yawning. In the cases with positive Wassermanns the hypertonia affected the entire body from the beginning, with a minimum in the face. The tremor was pronounced through the whole course of the disease and the two symptoms ran parallel throughout, from the beginning to end. Finally the tremor disappeared completely. In these cases there were no respiratory or other manifestations observed in the first group of cases.

The symptoms referable to the motor tract were similar in the positive and in the negative cases with the exception of the plantar reflex, which was negative at first and positive later in the negative cases, and *vice versa* in the positive cases. Also myoclonia was present in the negative cases and absent in the positive cases. The patellar tendon and Achilles tendon reflexes were increased in both groups of cases. Ankle-clonus was variable: no definite inference could be drawn.

In the domain of mental phenomena there was a manifest difference in the two groups of cases. In the positive cases there was only dullness, difficulty of concentration. In the negative cases there was no mental dullness but outbreaks of delirium with confusion lasting but a few days; they occurred at various times and parallel with elevation of temperature.

Conclusion. The foregoing analysis presents sufficient data for a differential diagnosis of encephalitis lethargica and syphilitic mesencephalitis. Without taking into consideration the cases which improved under antiluetic treatment although their Wassermanns were negative, the following important differential signs may be emphasized:

1. The Wassermann reaction.
2. The cytology of the spinal fluid. The marked diminution during the course of the disease of the number of lymphocytes favors encephalitis lethargica. Persistence of a large number of cells (although somewhat less than the original number) favors syphilis.
3. Late appearance but eventual disappearance of diplopia favors encephalitis. Early appearance and persistence of it point to syphilis.

The same may be said of ptosis and of Argyll-Robertson's pupillary reaction.

4. Among motor phenomena the presence of myoclonia is of some diagnostic value. It is usually absent in syphilis of mesencephalon but present in encephalitis.

5. Parkinson's syndrome may be present in both. In encephalitis the tremor is slight but persists indefinitely, while in syphilis it is marked and eventually disappears when the patient is under treatment.

6. Somnolence cannot serve a diagnostic purpose in the beginning, but later in the course of the disease it is protracted in encephalitis but not in syphilis.

7. Mental phenomena in encephalitis may be manifested in delirious or confusional outbreaks, but in syphilis there is only mental dullness.

8. Sphincters as a rule are not involved in encephalitis, but they are disturbed in syphilis.

9. Finally, fever is usually present in encephalitis, but absent in syphilis.

The following is a brief account of the cases:

Case I. Woman of twenty-five. Irresistible desire to sleep. Slight fever (99.4). Some headache. Facies set; features immobile, slight tremor of left hand. Knee-jerks plus on both sides. Achilles also plus; Wassermann negative in blood and spinal fluid. Lymphocytes 15. Delirious outbreaks of short duration. Diplopia and ptosis in the third week.

Course. Lymphocytes came down to six. Fever disappeared. Mental phenomena disappeared. Hypertonia and slight tremor remain after eighteen months. Eye symptoms totally disappeared. Slight somnolence still present.

Case II. Woman (single) of twenty-one. Headache, fever, (100). Myoclonia in upper extremities. Somnolence marked. Reflexes increased. Plantar reflex flexor. Some confusion. Hypertonia of face and fine tremor of right hand. Ptosis and diplopia on the twelfth day. Argyll-Robertson's pupil on the fifteenth day. Wassermann negative. Lymphocytes, 25 in the spinal fluid.

Course. Myoclonia and somnolence persisted for three months and then disappeared. Eye symptoms cleared up. Parkinson's syndrome persists after two years. Somnolence disappeared in the third month. Mentality normal. Lymphocytes 10.

Case III. Girl of sixteen. General malaise, temperature 99.4. Pain in the head of a neuralgic character. Somnolence slight. Hypertonia of the face; slight tremor of left hand. Myoclonic move-

ments in lower extremities; reflexes increased. Argyll-Robertson's pupil and diplopia on the tenth day. Wassermann negative. Lymphocytes 22.

Course. Eye phenomena disappeared on the twenty-eighth day. Somnolence subsided on the fiftieth day. Plantar reflex became extensor at the end of fifteen months. Parkinson's syndrome still persists. Lymphocytes 6.

Case IV. Girl of nineteen, after a brief period of general malaise with chills and fever of 99.4, developed a tendency to fall asleep several times a day. Bilateral ptosis on the tenth day. Increased reflexes with a flexor plantar reflex. Parkinsonian syndrome: the face is hypertonic and the left hand is animated with a fine tremor. Patient slightly delirious at times. Wassermann negative. Lymphocytes 30.

Course. All symptoms disappeared except the Parkinsonian syndrome. Lymphocytes 6.

Case V. Man of thirty, bookkeeper, became sad. In the evening of the same day he had a delirious outbreak with visual hallucinations. On the next day he became somnolent. He had a temperature of 99.5. The pupils were unequal. Diplopia and external strabismus appeared on the fifteenth day. Argyll-Robertson pupil was first observed on the twentieth day. Myoclonic movements were seen on the face the spinal fluid. Parkinson's syndrome slight.

Course. Ocular phenomena disappeared completely. Lymphocytes 10, and remained as such at the end of eighteen months. Somnolence is still present but greatly reduced. Parkinson's syndrome still persists.

Case VI. Woman of thirty-five, commenced to suffer from neuralgic pain in the head which lasted ten days. Soon she noticed double vision. Delirious attacks with hallucinations would occur in the evenings for several days; temperature 99.3. Somnolence made its appearance. Argyll-Robertson's pupil was seen on the twelfth day. Wassermann negative. Lymphocytes 15 in the spinal fluid. Reflexes increased. Hypertonia and slight tremor in the left arm and leg.

Course. Somnolence persisted for several months. Ocular phenomena disappeared. Lymphocytes 5. Tremor and fixed facies are persistent at the end of two years.

Case VII. Girl of twelve suddenly felt pain in the head and neck and continued so for fifteen days. She soon developed myoclonic movements of the abdomen, thorax, and diaphragm. On the third day the myoclonia invaded the lower extremities. At the same time somnolence made its appearance which existed only during the day. At night she could not sleep. There was a slight fever (99.3). The reflexes were increased, the toe phenomenon was flexor at first, but soon became extensor. Argyll-Robertson's pupil appeared on the

tenth day. Wassermann was negative. Lymphocytes 12. No Parkinsonian symptom-group.

Course. All the symptoms disappeared with the exception of the extensor plantar reflex, which in the eighteenth month still persists.

Case VIII. Man, thirty-two years old, commenced his encephalitis with diplopia. Ptosis and internal strabismus were seen on the left side. Somnolence appeared on the fourth day and it was slight. Insomnia at night was present. The reflexes were increased and the plantar reflex was flexor in type. Wassermann was negative. Lymphocytes 11 in the spinal fluid. Mild fixation of the face was evident but there was no tremor. No fever.

Course. The eye symptoms disappeared in two months. Somnolence in a very mild form persisted for eight weeks. Lymphocytes 5. Complete recovery.

Case IX. Man of forty-five, barber, was seized suddenly with vertigo, fever, and vomiting. On the third day these symptoms disappeared, but they left him in a state of exhaustion. He became drowsy, would frequently fall asleep. On the twelfth day Argyll-Robertson pupil was in evidence. The left knee-jerk was increased, ankle-clonus was on both sides and the plantar reflex was flexor in type. Hypertonia of the facies with a fine tremor of the left upper extremity was present. Wassermann was negative. Lymphocytes 16 in the spinal fluid.

Course. In the eleventh month after the onset, the eye symptoms and somnolence as well as the ankle-clonus disappeared. Curiously enough the left plantar reflex became extensor and remained persistent. The Parkinsonian syndrome is still present. Lymphocytic count came down to 5.

Case X. Man of thirty-eight, storekeeper, had been complaining for several days of neuralgic pain in the head accompanied by disturbed vision. In several days the pain subsided and the patient became somnolent. At night he had delirious outbreaks during the first week. Myoclonic movements soon appeared in the extremities and abdominal muscles. Ptosis, diplopia, myosis made their appearance on the tenth day. A slight tremor was seen in the left arm and leg. Wassermann was negative. The lymphocytic count in the spinal fluid was 18.

Course. In the eight weeks all the symptoms subsided considerably, the lymphocytes number now 6. The tremor is persistent, eighteen months after the onset.

Case XI. Woman of twenty-seven, bookkeeper, complained of general malaise, backache, and severe pain in the head. Soon she became drowsy, would fall asleep at any time during the day. Diplopia and external strabismus with nystagmiform movements of the eyeballs were then observed. Myoclonia was quite marked in

the upper extremities and face. Argyll-Robertson's pupils soon appeared. The knee-jerks were increased, ankle-clonus was present on both sides and the plantar reflex was slightly extensor on both sides. On the thirtieth day of the malady Parkinsonian syndrome began to be manifest: the face became fixed and a slight tremor appeared in the left hand. The Wassermann was negative. The lymphocytes in the spinal fluid numbered 35.

Course. The symptoms gradually subsided and on the fifth month all disappeared with the exception of the Parkinsonian symptoms which are still present. Lymphocytes are now only six in number.

Case XII. Girl of seventeen after a period of general malaise with a slight elevation of temperature (99.4) lasting several days developed diplopia and a tendency to fall asleep whether at the table or in company. On the twelfth day myoclonic movements appeared in the four extremities. The left knee-jerk was increased and the plantar reflex was extensor in type. The Wassermann was negative and the lymphocytes of the spinal fluid numbered 16. There were no eye symptoms with the exception of nystagmiform movements of some duration when the eyes were turned to the right.

Course. Gradually the symptoms became ameliorated and at the end of seventeen months totally disappeared. At the time the lymphocytes numbered 7.

Case XIII. Woman of thirty-five was seized with a chill, back-ache and unusually severe pain in the head of a neuralgic character, also fever 100. In three days the condition cleared up, but she became somnolent. Myosis and Argyll-Robertson pupils soon became noticeable. The knee-jerks were greatly diminished and an extensor plantar reflex was demonstrable on the right side. At the same time the patient had some difficulty of micturition. There was considerable mental hebetude. The Wassermann reaction was positive in both humors. The lymphocytic count was 145. At the end of six weeks Parkinsonian syndrome was in evidence: it was much more pronounced, the hypertonia was noticed over the entire body and a tremor of the right arm and leg was of a wide amplitude.

Course. The somnolence was marked the first two weeks and then gradually disappeared within the following three weeks. The eye symptoms persisted in spite of the antiluetic treatment. The reflexes at the end of eighteen months were found increased on both sides but the plantar reflex became normal. The state of the sphincters remained unaltered. The hypertonia persisted, but the tremor gradually disappeared. The lymphocytes were 75 and persisted.

Case XIV. Man of thirty-two, chauffeur, developed fever (100.1) and pain in the head. On the tenth day these symptoms disappeared but the patient was left in a state of asthenia. Soon somnolence made its appearance and it was very much pronounced.

Diplopia and nystagmus also ptosis were in evidence on the 24th day. The knee-jerks were exaggerated and the plantar reflex was extensor in type. Parkinsonian symptom-group was quite pronounced. Wassermann reaction was positive in both humors. Lymphocytes numbered 150.

Course. The somnolence lasted but three weeks. The state of reflexes remained unaltered at the end of fifteen months. The same may be said of the eye symptoms. The lymphocytes count was then 80 and persisted in spite of the treatment. The Parkinsonian syndrome eventually disappeared.

Case XV. A boy of nineteen, football player, developed suddenly general malaise, headache, and a slight fever (99.2). Rapidly intense somnolence developed which remained pronounced during a period of fifteen days. Parkinsonian syndrome made its appearance, the tremor was confined to the left arm and leg. Then strabismus, ptosis, Argyll-Robertson pupils appeared. The knee-jerks were unequal. The sphincters were involved. Mentally the patient was dull. The Wassermann reaction was positive in the spinal fluid. Lymphocytic count was 140.

Course. At the end of sixteen months the lymphocytes were 90 in number, the Parkinsonian symptoms were absent; the plantar reflex became flexor. The eye symptoms remained unaltered.

Case XVI. A girl of eighteen, clerk, complained of dim vision and headache. Rapidly somnolence set in. It was very much pronounced. At the same time there was fever of 100. Argyll-Robertson pupils were evident very early. When at the end of 12 days the somnolence became slight, Parkinsonian manifestations were noticed: the hypertonia was generalized and the tremor was gross on the left side. The reflexes were increased. Mental dullness was marked. Wassermann reaction was positive in the spinal fluid. Lymphocytes 135.

Course. The lymphocytes came down to 90 and later to 74 but since then there has been no change in the number. The Wassermann reaction remained positive at the end of two years in spite of the treatment. The state of reflexes, of sphincters, of the eyes remained unaltered. The Parkinsonism has totally disappeared.

Case XVII. Woman of forty-one presented apparently all the symptoms of influence. After all the acute symptoms have subsided, somnolence developed. It was so pronounced in the beginning that it was often difficult to awaken her for feeding. When at the end of ten days its intensity decreased, the eye symptoms became evident, namely, Argyll-Robertson pupils and diplopia. Soon a Parkinsonian facies was observed, but no tremor. The tendon reflexes were unequal and the plantar reflex was extensor on both sides. The sphincters were slightly involved. The Wassermann reaction of the spinal fluid alone was positive. There were at first 140 lymphocytes.

Course. After a prolonged treatment with antiluetic remedies the number of lymphocytes in the spinal fluid went down to 75 and remained as such at the end of seventeen months. The Wassermann remained positive. Mentality reflexes are the same, but the plantar reflex is now flexor. The Parkinsonian hypertonia disappeared completely. Argyll-Robertson showed no improvement but the ptosis disappeared.

Case XVIII. Woman of thirty-five, recovering from childbirth became somnolent in a most intense degree so that it became extremely difficult to arouse her. During that stage an examination revealed unequal extensor plantar reflex, occasional incontinence of the sphincters. A lumbar puncture showed a lymphocytosis of 150 cells and a positive Wassermann. When at the end of 15 days the somnolence considerably lessened, a bilateral ptosis was visible. Soon Argyll-Robertson pupils were observed. Headache was marked. Mental hebetude was marked. Hypertonia of the entire musculature and a unilateral tremor were pronounced.

Course. The lymphocytes were reduced to 78 and remained as such at the end of sixteen months. The Parkinsonian symptom-group disappeared. The sphincters improved. The eye symptoms remained unaltered in spite of a vigorous course of antiluetic treatment.

THE APPARENT EFFECT OF ARSPHENAMIN IN TWO CASES OF BRAIN TUMOR *

BY ANGUS W. MORRISON, M.D.

AND

J. CHARNLEY MCKINLEY, M.D.

MINNEAPOLIS, MINN.

Apparently little attention has been directed in this country, if the paucity of medical papers is any indication, to the cases of brain tumor which have been treated, whether purposely or under mistaken diagnoses, by arsphenamin. A few reports which have appeared in the German medical literature seem worthy of brief mention in connection with the two cases presented in this communication.

Czerny and Caan (1) treated a number of cases of inoperable malignancy of various regions of the body with salvarsan and thought they obtained relatively favorable symptomatic results, though naturally the patients were not cured. Most of their cases had positive Wassermann reactions so that it is possible some of the improvement was due to the treatment of a syphilitic process.

Zieler (2) treated a case of inoperable lymphosarcoma with salvarsan without any noticeable improvement. In a case of sarcoma of the neck (clinical diagnosis), the patient developed severe pain, swelling and softening of the tumor following two injections. The tumor then began to grow more rapidly and the patient died two months later.

Noehte (3) treated a case of malignancy with metastases in the brain, producing temporary improvement. However, the patient gave a history of syphilis.

An angiosarcoma of the brain was treated with salvarsan under a diagnosis of syphilis by Jooss (4). The patient's symptoms were rapidly progressive a day after the second injection. Brain tumor was suspected and operation was performed immediately. The patient lived for three days following the operation. At autopsy a tumor was found which was undergoing liquefaction. Jooss thinks the liquefaction was the result of the action of the salvarsan.

Matzdorff (5) observed temporary improvement in a case of

*From the Division of Nervous and Mental Diseases, University of Minnesota Medical School.

gliosarcoma of the brain under treatment with salvarsan. This patient denied any venereal disease and the spinal fluid reactions were normal on repeated examinations except for a single positive Wassermann reaction.

It is the opinion of the above observers that salvarsan frequently affects the course of a tumor, either favorably or adversely, depending upon the results obtained in their individual cases.

CASES

Neuropathology No. 221—male—age thirty-three. Patient was first admitted to the University Hospital April 18, 1919, complaining of headache, loss of vision, intermittent anesthesia of the right half of the face and right arm, and dizzy spells. Family history was unimportant. Patient was a carpenter. Had diphtheria at four years of age; measles at nine. His left eye was injured at seven years of age and he was blind for four years at that time. Following this there was some improvement but twelve years before admission he developed a strabismus. Two years before admission the patient was struck in the left eye by a cold bolt but he has had no trouble with this eye since that time.

Present Illness: For two years before admission to hospital, patient had headache which was sharp in character and continuous day and night, lasting as long as a week at a time with a few days free interval. The pain was situated above the eyes and usually was accompanied by dizziness. Headaches became gradually more severe and persistent. The scalp was hyperesthetic during an attack. There was a progressive failure of vision. Only on two occasions did patient vomit with these headaches. For a few months before admission he had been having occasional attacks of unconsciousness lasting as long as three days. There was no history of any motor disturbances but the right hand at times felt dead.

General physical examination was negative. Blood pressure, 130/90. Neurological examination—Ophthalmoscopic examination showed papilledema with hemorrhages in both eyes, especially the left. Both pupils were irregular, the right larger than the left. The latter reacted very sluggishly and incompletely to light and accommodation. The right did not react to either light or accommodation. There was an external strabismus of the left eye and a nystagmoid movement on looking to the right. Occasionally there was some tremor around the eyes and mouth. The other cranial nerves were normal. The deep reflexes of the upper extremities were active, possibly greater on the left than on the right. The abdominal reflexes were active on both sides, slightly more on the left than on the right. The knee jerks were not obtained except on reinforcement and then were elicited only with difficulty. The ankle jerks were also markedly reduced. There was slight incoördination on the left on finger-nose test and this incoördination was well marked on the finger-finger test. There was no evidence of any incoördination of

the lower extremities. Romberg was negative. No paralysis or paresis. No sensory disturbances found except for loss of vibration sense over the toes and a reduction over the bony processes of the lower extremities.

Laboratory findings—Examination of urine, blood and blood Wassermann were all negative. Spinal fluid—None, negative; 1 cell per c.mm.; colloidal gold and Wassermann reactions negative.

X-ray of skull showed no evidence of increased intracranial pressure and the sella was normal.

Patient was discharged at his own request eight days after admission while still under observation.

The diagnosis was indeterminate at this time but brain tumor (possibly gumma) or neurosyphilis was considered.

On April 20, 1920, patient was re-admitted to the hospital. In the interim he had been at work until November, 1919, when he stopped on account of weakness, staggering in the dark and because his feet dragged. His vision had continued to fail and for six months he had been unable to read. The spells of unconsciousness had become more frequent, occurring at least twice a week and sometimes daily. During the previous three months there was progressive impairment of memory. For six weeks before admission he was unable to walk, had been confined to bed, and had become progressively more stuporous. He was unable to feed himself and there was incontinence of bowels and urine, and he had some hallucinations. He also apparently had some difficulty in chewing and would either forget to chew or would forget to swallow the last mouthful. For four weeks before admission was disoriented. Bedsores developed over the sacrum and the left heel. His strabismus had been less marked for four months but became more evident during his spells of unconsciousness. There was no projectile vomiting. The left side of the jaw had become a little weaker than the right and there had been considerable loss of weight.

Physical examination was essentially negative except for decubitus ulcers over the sacrum and left heel. Blood pressure, 138/104. Neurological examination—patient appeared dull and stupid. Had no initiative; answered a few questions after a long pause; spoke in a slow, monotonous tone. Appeared tired and drowsy. Answers were foolish at times. Did not recognize the physicians who cared for him on his former admission and could not remember faces from day to day. Was disoriented as to time and place. He could not turn over in bed on account of weakness. Pupils as on previous examination. There was a slow nystagmus on looking laterally. There was a tremor of the right side of the lips. The right biceps reflexes were very active followed by a clonic movement of the arm. The left biceps was normal. Knee jerks and ankle jerks were markedly reduced even on reinforcement. Babinski reflexes were normal. A bilateral ankle clonus was present which was more marked on the left. There was a more or less constant fine movement of the right big toe, rhythmic in character and occasionally a coarse rhythmic

tremor of the right hand on active or passive movement. At times the right arm and leg were spastic.

X-ray of the skull April 29, 1920, showed some indefinite sella changes. Repeated urine examinations were negative except for occasional granular and hyalin casts. The blood was negative except for a moderate anemia. Blood sugar, 0.155; creatinin, 1.2; urea nitrogen, 13.485. Blood Wassermann positive. Spinal fluid examination, April 28—Nonne, negative; 1 cell per c.mm.; colloidal gold negative; Wassermann positive.

The diagnosis was still considered indeterminate but neurosyphilis was strongly suspected. Patient was apparently in extremis and an immediate course of anti-syphilitic treatment was instituted. He received four doses of 0.45 gram and seven doses of 0.6 gram of neo-arsphenamin at intervals of six or seven days. General condition remained about the same until the middle of May except for being a little brighter mentally. The bed sores had become more marked. Toward the latter part of May the patient became much brighter mentally but he was still somewhat confused and occasionally expressed abnormal ideas. At this time the bed sores began to improve rapidly. By the middle of July the patient was up and around. His mental status was almost normal three months after second admission and his improvement was so marked that his general condition was hardly comparable with that of three months previous. The sacral bed sore then measured only 1 cm. x 3 cm., whereas it was previously 15 cm. x 10 cm. The patient was discharged July 24, 1920, and, owing to his marvelous improvement under anti-syphilitic treatment, a diagnosis of neurosyphilis was made.

The patient was again admitted to the hospital December 2, 1920. Since his previous discharge he had not had such severe headaches but still at times had attacks of unconsciousness. He had been able to get around much better and had been working off and on at his trade as carpenter for four months. He had continued his anti-syphilitic treatments and had received eleven injections of neo-arsphenamin and one of mercury salicylate. Three weeks before the last admission his headaches again became severe and his eyesight much worse.

Examination showed little change in the patient's general status as compared with his condition on his second discharge. The blood Wassermann was negative and the spinal fluid negative throughout. X-ray of the head showed absence of the posterior clinoid processes. Ophthalmoscopic examination showed secondary optic atrophy. On the basis of the definite X-ray findings, a diagnosis of tumor, possibly pituitary, was made but an exploratory operation failed to reveal this. A few days after the operation, patient developed signs of meningitis and died.

At autopsy a cyst 5 cm. in diameter was found in the right frontal region of the brain. The wall of the cyst varied from 0.5 cm. to 1 cm. in thickness and on microscopic examination proved to be definitely gliomatous (Figs. 1 and 2).

The other autopsy diagnoses of significance were meningitis, flattening of the sella turcica and purulent bronchitis. No evidence of syphilis, either grossly or microscopically, was found in any part of the body.

The *second case* is Neuropathology No. 284, a woman thirty-seven years old—nurse by profession.

Patient was first seen in private practice in July, 1921. She stated that she had been exposed to syphilis four years previously while taking care of a luetic patient. At that time she developed a sore at the left angle of her mouth and was given one intravenous



FIG. 1. Section of the wall of the gliomatous cyst in Case 221. Hematoxylin and eosin. X 80.

treatment which caused considerable scarring at the site of the injection. Except for this the past history was unimportant.

Present illness: About a year ago she began to have attacks occurring at intervals and lasting two or three days with a few days free interval. These were preceded by dizziness and accompanied by nausea, vomiting, and flashes of light. There was some failing of vision. Patient also had periods during which she was speechless and had complete loss of memory. These periods were not associated with headaches and resembled petit mal attacks. She had no pains or numbness. For three or four months had had amenorrhea. There had been considerable loss of weight and loss of appetite. Her friends had noticed that there was some change in her disposition of late; that she was careless in her personal appearance, had a shuffling gait, used poor English, had a mild sort of conceit, being overpleased

because a dog was friendly to her, and the like, and she was no longer able to embroider.

Physical examination was practically negative. Neurological examination showed slight inequality of pupils which were a little irregular in outline. Both contracted through a small arc, quickly dilating. Ophthalmoscopic examination showed that the right nasal edge of the disk was indistinct and that there was a left optic neuritis. There was a moderate coarse tremor of the extended hands, more evident on the right. No paralysis. No paresis. Coördination good. Sensation normal. Arm jerks exaggerated but symmetrical.

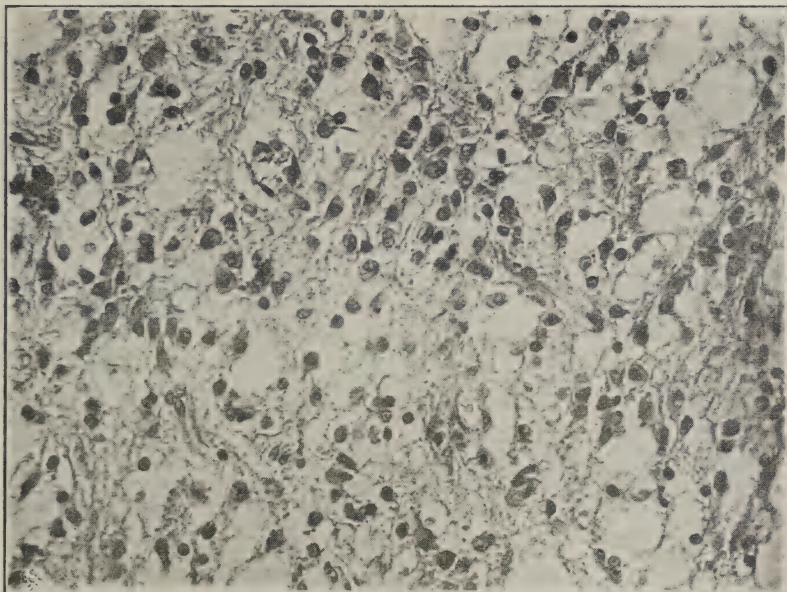


FIG. 2. Section of the wall of the gliomatous cyst in Case 221. Hematoxylin and eosin. X 300.

Knee jerks markedly exaggerated, the right possibly more than the left. Normal Babinski responses. Mentally the patient showed a condition similar to that brought out in her history, being somewhat dull and apathetic.

Laboratory findings: Urinalysis is negative. Blood examination negative. Blood Wassermann was negative with the ordinary antigens but ++ with the cholesterinized antigen. Spinal fluid was not under increased pressure; negative Nonne; 4 cells per cmm.; Wassermann negative.

The diagnosis of brain tumor was seriously considered in this case but in view of the rather definite history of exposure, it was thought advisable to try anti-luetic treatment. Patient received mercury rubs for five weeks with six intravenous neo-arsphenamin injections. She felt brighter, more cheerful, was rational, seldom

had dizzy spells; memory was greatly improved; headaches much better. She desired to return to work, her only complaint being that about once a day she had "little periods of losing herself." She was started on her second course of intravenous injections, receiving two. Her third treatment was omitted owing to a few hyalin casts being found in her urine. On the following day, November 11, 1921, patient had a sudden convulsion and died.

Postmortem examination showed practically no fluid in the pia-arachnoid spaces. The convolutions of the brain were markedly flattened and pressed together. The whole of the left temporal lobe consisted of a soft, flabby material which on section showed



FIG. 3. Section of the glioma in Case 284. Hematoxylin and eosin. X 80.

numerous areas of liquefaction. The color of this tumor mass was somewhat more yellowish than the normal brain substance. The temporal lobe had undergone a diffuse enlargement and had pushed into the brain substance so as to displace the basal nuclei medially. The general topography of the convolutions and sulci of the temporal lobe was preserved. There was a moderate internal hydrocephalus. No evidence of syphilis was found anatomically.

Pathologic diagnosis was glioma of brain (see Fig. 3 and Fig. 4).

Discussion: In the two cases presented above we have been dealing with gliomata treated with neo-arsphenamin under a tentative diagnosis of neurosyphilis. In both cases brain tumor had been very seriously considered but the improvement was so marked under anti-

syphilitic treatment that further therapy along these lines appeared indicated. Any opinion as to the action of neo-arsphenamin modifying the course of the development of the gliomata or changing the intracranial pressure produced by them would be purely conjectural. It is decidedly obscure from a pathological or physiological standpoint as to what occurred during the remissions the patients experienced. Further observation and experimentation along this line would be desirable. Interest in these cases lies largely in the fact that a therapeutic test for syphilis may lead one far afield from a true diagnosis. They further emphasize the need for caution in diagnosing neurosyphilis without positive spinal fluid findings of syphilis.

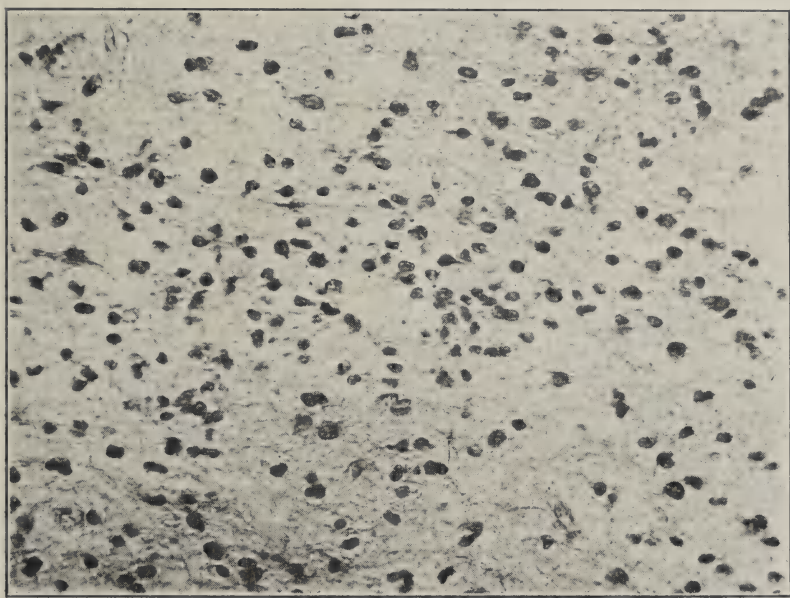


FIG. 4. Section of the glioma in Case 284. Hematoxylin and eosin. X 300.

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EPIDEMIC (LETHARGIC) ENCEPHALITIS *

A CLINICAL STUDY OF THIRTY-FIVE CASES GATHERED DURING THE RECENT EPIDEMIC, INCLUDING SOME RETROSPECTIVE CASES

BY MAX H. WEINBERG, M.D.

ASSISTANT NEUROLOGIST, WESTERN PENNSYLVANIA HOSPITAL,
PITTSBURGH, PA.

(Continued from page 146)

Case 3. Practically panophthalmitis of left eye, periods of stupor, severe headaches, mild changes in fundi, pyramidal tract involvement—resembling cerebral neoplasm.

M. B., No. 6404; American; age fifty-four; housewife. Admitted 2/16/20. Disch. 2/28/20.

F. H.—Mother died at the age of sixty-three—diabetes. Father died at eighty-one—apoplexy. Seven sisters living and well. One sister died at age of twenty-four—typhoid fever.

P. H.—She had the usual diseases of childhood. Pneumonia several times during early life. She has been deaf for twenty-eight years. The deafness came on very suddenly, following a sneeze, without any apparent inflammation of the ear drum, and there was no discharge at any time. She has two living children, thirty and thirty-two years of age. Has had several miscarriages since those births. Had influenza in 1918. Has suffered with ear trouble at various intervals since that time. This quieted down upon resting in bed.

The patient was first seen by her family physician 1/13/20. He found her in stupor. She was aroused with difficulty. Her breathing was stertorous. She answered questions with great difficulty, dropping off to sleep immediately. The chief complaint was headache and pain in her left eye. The left pupil was slightly larger than the right, and the least bit irregular. The ankles were slightly swollen and pitted on pressure. All other organs were negative.

The history showed that she had had headache almost constantly for two months, being much worse at times of exertion or excitement. The urine was negative at that time. She responded to treatment, but on January 26 she again went into a stupor bordering on coma, and this lasted for three days. Her only complaint on waking was intense pain in the left eyeball radiating to the left side. Uremia treatment brought some improvement but no let-up in the headache. Her headache was fairly constant, but she had paroxysms which lasted from fifteen to thirty minutes.

* From the service of Dr. W. K. Walker.

On February 3, ophthalmoscopic examination showed a marked choked disc in the left eye, and beginning choked disc in the right.

The doctor in his communication adds "that he hands her over with the belief that she has a brain tumor. She seems better than she was, but does not gain strength, and is more or less stupid all the time, sleeping night and day except when aroused and talked to. Her station is not good and her reflexes are almost minus. Her mental condition is not as good as it was. Her speech is thick. She is forgetting how to say her words, and her tongue seems thick. Her memory for past events is very poor. Ptosis of the left eyelid came on February 1. Diplopia came on a few days preceding this date. Her vision is not as good as when she first took sick."

2/16/20. On admission the following notes were made: Complete ptosis of the left eyelid. Left pupil completely dilated. No reaction of the left pupil to light. Complete paralysis of the third and fourth nerves on left. Suggestive paralysis of the left sixth. Slight nystagmoid movement on left. Left fifth nerve involved. There is corneal anesthesia on the left side. Margins obliterated in both fundi. Grip in both hands good. Arm reflexes present, slightly increased on the right. Knee jerks diminished on the right, achilles normal. No Babinski on right. No ankle clonus. Left knee jerk diminished, achilles normal. Babinski present on left. No aphasia or astereognosis. No mental symptoms.

The following note was made on the next day: Owing to fleeting nature of the symptoms, an increase of cells of the spinal fluid, especially lymphocytes, and nature of semi-conscious state, the diagnosis of epidemic (lethargic) encephalitis is suggested.

Eye examination—Dr. Carson. Left pupil dilated and globe immobile, except for some action of the external rectus. Both fundi are negative for any gross abnormalities, though caliber of arteries in both eyes seems slightly reduced. Both discs perhaps slightly pale, but this is not marked, and discs appear the same on the two sides. Diagnosis of fundus condition—negative, except for slight evidence of sclerosis.

The subsequent course of the disease proved it to be definitely a case of encephalitis. The patient stayed in the hospital but a short time and was observed for a while at her sister's home. The symptoms subsided gradually, and when she left the city there were still many residual symptoms.

Temperature on three separate days was 99. Pulse was never higher than 100, and usually between 84 and 90. Respiration normal.

Case 4. Myoclonic movements of the left abdominal muscles, radicular pains, and slight paresis.

E. C. Age fifty; Englishman; No. 6305. Admitted 2/9/20. Dischg. 2/28/20.

F. H.—Wife living and well. No children. No T.B. in family. Habits, good.

C. C.—Pain in left side, kidney region.

About seven days before admission, the patient came home with

sick headache. It lasted two days. On the very day that the headache improved, pain began in the left side. This pain was throbbing and jumping over the left kidney, and did not radiate up or down, but seemed to radiate antero-posteriorly. The pain was so severe at times that it made him cry out, but it did not double him up. It required morphine to quiet the pain. The next day there was a twitching of the muscles of the left abdomen. This, however, seems to have been present ever since the pain started.

2/16/20. The first neurological examination was done on this day and the following notes made: The pupils are irregular, react to light, but do not hold well. Consensual reflex and accommodation normal. Fundi normal. Slight deviation of the tongue to the right. Facial folds somewhat obliterated. Apparent weakness of the left face. No tremors. Rigidity in both arms so marked that reflexes cannot be taken. Grip in both arms good. Abdominal reflexes active. Cremasteric reflexes present. Knee jerk normal on right, right achilles not elicited. No Babinski or ankle clonus. Knee jerk somewhat increased on left, and achilles absent. No plantar flexion of the great toe on stimulation. Myoclonic twitchings of the left abdominal muscles are rather pronounced.

2/22/20. The subjective signs are the same. The patient states that he has developed a ravenous appetite for the last few days.

The temperature on admission was 100, and during the first eight days was around 99, after that normal. Pulse between 90 and 80.

Reëxamination 4/2/22. The patient went to work at his trade a few weeks after he left the hospital. He is as capable as he ever was at his work. He complains of a slight pain in the left upper gluteal region which came on just before he left the hospital. It comes on only in the morning. He had nocturia, once or twice during the night. He describes it as inability to hold his water. He noticed that he does not care for sexual relations. His wife was operated on and he abstained for the last two years without being disturbed by it.

Appearance good. Pupils dilated, both irregular, the left more so. Both pupils react well to all stimuli. Ocular movements normal. The nose only shows distinct greasiness. Tongue tremulous, slightly deviated to right and shows evidence of mild atrophy on right. Slight weakness of left face is only suggestive. No tremor of hands. Arm reflexes slightly diminished. Right knee jerk somewhat increased, left is normal. Achilles normal. No Babinski or ankle clonus. No sensory disturbances.

No psychic changes, with the exception that he cannot concentrate well. When he listens to a sermon, his mind wanders off.

Case 5. Restlessness at onset, followed by lethargy and stupor. Catatonic rigidity of arms and hypotonia of hand. Parkinsonian syndrome, and fairly marked psychic disturbances.

J. H. Age forty-three; American; painter; No. 6881. Admitted 3/9/20. Discharged 5/28/20. F. H.—Father and mother living

and well. Two brothers and three sisters living and well. one brother and one sister dead—cause unknown. P. H.—Diphtheria thirty years ago. Colic ten years ago. Denies venereal infection. Habits—Uses tobacco moderately. C. C.—Pain in the head over each eye. Dizziness.

The onset of the disease took place two weeks before admission to the hospital with a dull frontal headache, and slight dizziness. The dizziness increased gradually in severity but the patient kept at his work for four days. He could not sleep at night. On the fifth day he stayed in bed, and since then he has been in and out of bed. He could not stay in bed constantly owing to nervousness and restlessness. His eyesight was poor, and he could not read because the print would become very small. For the past four days he had poor appetite. He has also been troubled with oliguria. For the last week he has not been able to walk steadily in the light and he staggers in the dark.

3/12/20. Neurological examination—The patient is in stupor. He does not respond when talked to. The face seems to express pain. The eyes are tightly shut and resist an effort on the part of the examiner to open them. When the eyes were partly opened, the pupils were found dilated. The right does not respond to light; the left has a faint reaction to light. Nystagmus present. Patient refuses to open the mouth. There is rigidity of the neck. Face greasy. Both arms are very rigid. All the deep reflexes are fairly active, but the right achilles is markedly increased. Catatonia not very marked. No Kernig.

The next day the patient coöperated readily and responded to questions very well. There was a definite tremor of the Parkinsonian type, and the rigidity was still marked. The tongue was very tremulous. The knee jerks and achilles were increased, but the right achilles more so. There was a peculiar waxy yellow appearance of both lower extremities.

Two days later the patient was still stuporous. Hypotonia of the left hand was noted, and the right arm was rigid. Both knee jerks were absent and both achilles were increased.

The patient's stupor kept up for another week. The reflexes varied, and outside of the hypotonia of the left hand, the rest of the musculature was very rigid. Characteristic Parkinsonian tremor, especially in the right, persisted. Catatonia was very marked. The patient ran a protracted course with but few changes in the neurological picture. He became markedly emaciated. The stuporous and lethargic state lasted for about four weeks.

The temperature on admission was 99.2, and stayed around 99 for some time, but on 3/30/20, it took a sharp rise to 102.5 and receded the next morning to normal. It again rose to 99 for about a week, after which it remained normal. The pulse at first was as high as 110, and fluctuated down to 80. Usually the pulse went along with the temperature. During the second week, the respirations were up to 30, otherwise they were normal.

Reëxamination. 3/26/22. The patient states that he felt bad for three months after his discharge from the hospital, and that he did not go back to work until April, 1921, about one year after his discharge. He complains of an occasional nervousness in the eyes, but cannot give any details.

The patient presents a stiff appearance, a slightly masked face, and he has a definitely slow speech, which is somewhat infantile. The right pupil is very irregular and larger than the left. It reacts very sluggishly to light and sympathetic stimulation, but is normal to accommodation and consensual. The left pupil is normal. Ocular movements normal. Tongue very tremulous, but in mid-line. No facial palsy. Moderate tremor of the right hand only. Deep reflexes of both arms slightly increased. Right knee jerk increased, left knee jerk normal. Right achilles normal, left achilles very much increased. No Babinski or ankle clonus. No rigidity of arms, slight rigidity of neck. No sensory disturbances. No psychic changes noted. He gets along at his work as well as he did before he took sick. He is doing the same work and his efficiency has not been interfered with in any way.

Case 6. Confusion, marked rash, monoplegia, unilateral hyperidrosis, and clonic movements of fingers synchronous with respiration. J. Y.; age thirty-four; married; Slav; laborer; No. 7130. Admitted 3/18/20. Discharged 7/6/20. F. H.—Cannot be obtained. P. H.—Negative. Habits—No alcohol or tobacco used by patient. C. C.—Nervousness, insomnia, pain in arms, chest, and shoulders, hallucinations, and difficult micturition.

Onset of the disease took place four weeks before admission with what the patient calls "the shakes." At first the doctor diagnosed the condition as "flu," but he has continued to be nervous and shaky since that time. At the present time the patient is having visual hallucinations. He sees a great many people, particularly soldiers, passing before him and trying to play tricks on him. They never attempt harm.

3/19/20. The patient is confused and disoriented in all spheres. The pupils are irregular and react to light sluggishly. Ocular movements normal. Von Graefe positive. Facial muscles very tremulous. Thyroid somewhat enlarged. Rose spots over abdomen and legs. The spots disappear on pressure.

3/20/20. The patient is quite toxic in appearance. The general tremor still persists. Pupils practically stationary to light. Right tonsil enlarged and diseased. There is a macular and mottled erythematous rash, particularly on the chest, but also to a lesser degree upon the arms and head. Desquamation is also present. The reflexes are not altered.

Two days later, additional neurological changes were noted. The patient was somewhat confused and lethargic. The general tremulousness was not so marked. The pupils reacted to light sluggishly. There was slight weakness of the facial muscles. The tongue devi-

ated to the left slightly. Both knee jerks and right elbow jerk were absent, and the left elbow jerk was diminished. The skin was very moist, the rash disappeared completely. On this day the diagnosis of encephalitis was ventured.

About a week later, the patient had increased respiration, and a rhythmic movement of the fingers of the right hand coinciding with respiration was noted. Profuse sweating came on several days later. This was limited to the left half of the body.

The patient developed paralysis of the right arm, and for the first time had a Babinski sign on the left.

5/10/20. Again there developed a rash over the abdomen and thighs. Movement returned to the right arm. He complained of abdominal pain. Middle fingers of the right hand still moved synchronously with respiration, and the sweating of the left half of the body still kept up. During the next six weeks of the patient's stay in the hospital there was practically no change except for the development of cephaloptosis.

The temperature was at 100 for first four weeks, then around 99 for the rest of the stay. Pulse around 120 and down to 90. Respiration mostly around 30, and occasionally would go down to as low as 24.

Case 7. Convulsions, crossed paralysis-Millard-Gubler syndrome, cerebellar symptoms, and euphoria. H. McC.; age twenty-seven; American; laborer. Admitted 3/20/20. Discharged 5/22/20. F. H.—Negative. P. H.—Negative. Habits—No information can be obtained. C. C.—Lethargy, diplopia, and paralysis.

About six weeks before admission the patient had what was diagnosed as the "flu," from which he did not recover fully. Then he developed what was called "tonsilitis," and after the tonsilitis attack he complained of diplopia, and slept a great deal. He could only be aroused for his meals. He stopped talking four days before admission.

Neurological examination—The patient looks very ill. The complexion is anemic and livid. The eyes are half open. Pupils somewhat dilated, but they react to light well. Ocular movements cannot be tested. The tongue protrudes with difficulty, apparently in midline. Facial fold obliterated on left side. Slight rigidity of neck. Right arm is weak and hypotonic. Hypotonia and tremor of left hand. Biceps jerks are exaggerated. Abdominal reflexes present. Both knee jerks are increased, right more than left. Ankle clonus present on right. Fundi normal. No retinal hemorrhages noted.

3/20/20. Paralysis less marked and the patient coöperates readily.

3/22/20. Weakness developed in left arm. The patient shows a tendency to laugh unduly. The right facial palsy is more marked.

3/25/20. The patient developed typical Parkinsonian position of the right hand.

3/31/20. Intention tremor of left hand very marked.

4/4/20. Both external recti are paretic.

4/9/20. The patient is euphoric. He laughs most of the time. Pupils unequal, left greater than right. The right reacts well, the left reacts sluggishly. Power in the right arm is returning. He can hold up his arm for a minute or so. The deep reflexes of the right arm are increased. There is ataxia in the right leg.

4/22/20. Facial weakness cleared up. Pupils react to light. There is much more power in the right arm. He is still unable to raise arm above shoulder. Intention tremor still marked on left. *Adiadochokinesis* present on the left. Ankle clonus, patellar clonus, and Babinski present on right. Left knee jerk practically normal.

Temperature during first week between 99 and 100, after that mostly normal. Pulse never over 100, and respiration normal after the first two weeks.

Reëxamination. 4/27/22. The patient's speech is slow, drawn-out, and monotonous. He is rather self-satisfied with his condition. He has been under the care of a policeman-chiropractor for a year and he has faith that this man will cure him.

The lines in his face are very deep and there is no semblance of masked face. The right hand is held flexed at the metacarpophalangeal joints, and the fingers are rigidly extended. The thumb is closely adducted. The eyes are normal. There is some overaction of the right face. The jaw deviates to right definitely. No tremor of hands. The right arm is held in pronation and at an angle of thirty degrees at the elbow. The wrist is everted. The tonus of the hand muscles is very much diminished.

All the arm reflexes, especially on the right, are markedly increased. Knee jerks are increased, especially on the right. There is a crossed knee jerk reflex, left to right. Achilles on left normal, on right increased. There is ankle clonus on right. Marked Babinski on right. Abdominal reflexes diminished on right, normal on left. Ataxia of left hand—finger to nose test. No sensory disturbances. Gait is hemiplegic in character. The right leg is flexed at the knee. He steps on the toes only, the heel being retracted. On walking, the left half of the body is pushed forward, the right leg making a sort of semicircle in bringing up the rear.

There is a movement of the right arm including the three outer fingers of the right hand which reminds one of the movements of *dystonia musculorum*. This occurs about once every ten seconds.

The patient is markedly euphoric. He has no insight into the seriousness of his condition. He laughs in a silly manner. His wife reports that he is very restless and shiftless. He stays in the house for his meals only. He had secured work as a watchman, but gave it up promptly.

I learned later that he threatened to kill his wife and that he was committed to the psychopathic ward of the City Hospital.

Case 8. Choreiform movements, early lethargy followed by sleeplessness, mild Parkinsonian symptoms, labial paralysis, and spasmodic tic of left arm and face: A. C.; age eighteen; shoemaker; Jewish; No. 7285. Admitted 3/24/20. Discharged 6/6/20. P. H.—Influ-

enza, pneumonia, and empyema one year ago. Denies venereal infection. Habits—Good. C. C.—Double vision and purposeless movements.

Five days before admission, after a hard day's work, he felt sleepy, but after conversing a while was aroused and could not sleep for the next four nights. His mother also noted that he was talking incessantly. The very next day he developed diplopia which persisted and on the next day developed choreiform movements.

Physical examination—During the examination the patient had been performing continuous movements of the arms and hands of a choreiform type. The patient is well oriented. Speech is clear and well ordered, but there seems to be additional exertion in uttering his words. The skin is hot and dry. The right pupil is larger than the left and does not react as readily to light. Both pupils react to accommodation. There is slight weakness of the right internal rectus on converging. No other muscular defects. Diplopia is manifest, but after a few seconds single vision returns.

The facial expression is of an ironed out type. There is, however, no weakness of the facial muscles. Tongue protrudes in mid-line, is heavily coated. Tonsils enlarged and infected. The thyroid is not palpable. The heart is negative. Biceps jerks diminished. Coarse tremor of hands. Left knee jerk is diminished. No ankle clonus or Babinski. During the latter half of the examination, the patient has been delirious. Sings and talks of the shoemaker's trade.

3/21/20. Eyes very restless. Constant grimacing of the face. The patient is very lethargic. Right eye partly open. Left eye closed. Face flushed. Knee jerks increased. Achilles suppressed. The patient is confused and disoriented.

3/24/20. The patient complains of pain in back of the head and eyes.

4/4/20. Tongue deviates to the right. Slight weakness of the left face. All the deep reflexes are suppressed. The patient is very lethargic and has to be aroused for his meals.

4/9/20. The patient developed typical mask face. Pupils unequal. Desquamation of face and lower part of trunk noted. Knee jerks and achilles increased. Lethargy still marked.

4/12/20. The patient is unable to move his lips and articulate properly. He is unable to whistle or move his lips to show his teeth. Also complains of dysphagia.

4/15/20. Lips are still paralyzed. The patient complains of excess of saliva which he cannot bring up. His articulation is so flat that one can hardly make out what he says.

4/16/20. The patient cannot move any muscle of the face. The eyes can only be closed partly. Sensation of face good. He cannot move eyebrows, lips, or nasal muscles. Paralysis of facial muscles began in present marked way with upper lip. Labial paralysis somewhat improved.

5/3/20. A note by Dr. Wholey reveals that the patient does

not remember his first week's stay in the hospital; that he has felt well all the time, and that he had funny dreams at first.

6/4/20. For the last two days he developed spasmodic tic of the left face involving the labial muscles and sternocleidomastoid muscle. Spasm occurs about one every minute. He has had pains in the neck for the last few days.

Temperature on admission 102.6, then dropped to 101, then fluctuated between that and 100, then normal. Pulse between 120 and 80 for the first two weeks, then normal.

Reëxamination. 11/21/20. Masked face fairly marked. Tongue is deviated to right. Spasmodic tic of left side of body is present, more marked in shoulder and facial muscles. There is fibrillation of the muscles of the left face. All the deep reflexes are increased.

During the last month he has had periods of unconsciousness lasting about five minutes. They occur about twice a week. He never bit his tongue, nor did he have involuntary urination during these attacks.

(*To be continued*)

TRANSLATIONS

EMOTION, MORALITY, AND BRAIN*

BY PROF. C. V. MONAKOW

ZÜRICH

(*Concluded from page 156*)

Now from the physiological-anatomical viewpoint, one must remember that really all visceral ganglia are connected more or less closely, that is, are related to one another, even in so far as their spatially closely outlined, cortical or subcortical representation is concerned; then too in the important reciprocal relations between the single glands (for instance, between the thyroid gland and the adrenals, the sexual glands and the chromaffine system, and so on). According to my experience a chemistry related to the inner secretion could represent the connecting link here. Considered more broadly it may be recalled, that for instance the vagus is responsible for the innervation of the thyroid as well as for that of the heart and of the digestive organs, and that inhibitions or stimuli in a peripheral innervation sphere of the oblongata, or higher upwards, can go out from a definite place to various areas. I do not assume here as the determining factor a true "conversion," a psychic repression of emotionally toned complexes in somatic effects (In Freud's sense). The "somatic" processes, which are exhibited in unresolved pathological conflicts, are coördinated (not subordinated) with the harmful experience, which has been temporarily carried over into the world of oblivion; in my opinion both phenomena present defense reactions biologically parallel and equal in value to one another only existing in different spheres.

And does pathological anatomy play no part in injuries to the world of emotion and morality? Of the pathological processes only inflammation, or infection accompanied by radical changes in the blood chemistry, and spreading diffusely in the cortex (for instance, meningitis), can produce profound changes in the emotional sphere (conditions of delirium, repression, exaltation). Poisons which cause continued disturbance in the inner secretions might produce similar results (for instance, Basedow's disease). But never, in my

* Authorized translation by Gertrude Barnes, A.B., and Smith Ely Jelliffe, M.D., of the authors Gefühl, Gesittung und Gehirn.

opinion, do emotional injuries spring up on a true morphological-anatomical basis, for instance as result of a hemorrhagic lesion or of other diffuse, gross changes in the cortex. The last have only dementia or disturbances of orientation as a consequence, not injuries in the sphere of emotion and affect. As is the case of underdevelopment or constitutional anomalies the majority of pathological processes in the cortex contribute to the pathology of the emotions only so far as they retract the natural discharge of emotionally toned conflicts (in the sense of psychic shocks) or as they produce a certain mental dulness.

I once read somewhere that grains of corn that were found in old Egyptian tombs, and that had lain hidden through thousands of years, when planted in fertile land, sprouted up, and produced ears of corn. The same thing happens *mutatis mutandis* to the emotional tensions (produced through strongly affect-laden experiences): stored in the human cortex, unreleased and long postponing their activity in an obscure latent state they can dwell there for years. From time to time they stir nevertheless (quickened by sympathetic associations), and cause even directly somatic disturbances in the startled host, especially if he is ignorant of their true relation with the incident experienced, and is surprised by allusions connected with the experience. That is to say, such inveterate psychic "tensions" ("complexes"), if touched by the right "key" (conversation, hypnosis, association tests) and brought to ekphory through symbols, that is, suddenly freed from "their captivity" (Freud's repression), then break forth anew. The passionate emotion repressed into oblivion, suddenly released, is again set free—sometimes with its elementary force and accompanied with all its separate visceral and bodily symptoms, and after such expression the patient may feel relieved by the discharge which is satisfying, though it has been delayed. But the contrary can occur (in unskillful medical conversation), and the "complex" laden with still severer suffering, may recede anew into the latent state. Herein it is a matter of indifference whether the psychic wound was evoked through a sexual or other insult (affecting self-preservation, honor, and so forth, and whether it originated in the earliest childhood or at a later time. A main condition for its taking place, is, however, that the episode serving as a basis for the psychic trauma was repressed into the latent state by a defensive act, that is, by a defensive act (designedly?) was "forgotten," and that later the possibility to conquer the insult in the unconscious was denied the individual.

Our knowledge of the so-called emotionally toned complex, of their "repression" (Freud) into the unconscious, and of their discharge, we owe above all to the works of the school of the so-called psychoanalysts (Breuer and Freud, Bleuler, Jung, Adler, and others). Through innumerable specific observations I have been able to confirm the correctness of many of the clinical facts taken as a basis for the explanations of those authors. The observations and conclusions of these authors are exceedingly valuable biologically, even if I cannot, to speak plainly, accept all their explanations without further confirmation. They tell us:

1. That the affects or conflicts once released in connection with insults to our "most important life interests" regress into a consequent latent state in the central nervous system in the sense of unresolved "tensions," if they do not find a rational, that is, harmonic discharge (or if a suitable abreaction at the time of the experience does not take place). Notwithstanding that the patient often cannot remember spontaneously the experience in question and the accompanying moments; they remain unforgotten and undischarged. These "retentions" now often (even in well people) cause nervous, somatic disorders, which usually occupy the foreground of the patient's complaints, and

2. That stimulating conditions laden with severe emotions existing in the latent state, and ideas connected with them are still in the position after years—if they again come into actuality through associative paths—to set functioning once again the inner secretions, or, the visceral nerves, as they did in the past experience, that is, to permit the stimulation complexes involved to be set acutely "in motion," just as were the "old Egyptian grains of corn in a fertile field." There lives latent in the "complex," the "force" to become actual, to evoke the same "chemism" in our blood, or to change temporarily, while in the latent stadium the complex only had the power of causing uncontrollable discomfort and periodical manifestations in the form of "attacks." Perhaps the two systems—the sympathetic and the autonomic—even with reference to their higher representatives in the cortex, are antagonistic to one another ("representations in the latent stadium"). At any rate, many experiences confirm the view that there is a close relationship between the emotionally toned events and inner secretion, and it is very probable that there is a very considerable participation of biochemical moments in the production of emotions.

And now we must turn to the dissolution, that is to say to the acute (temporary), and to the progressive "liquidation," of morality.

DISSOLUTION AND "LIQUIDATION" OF MORALITY

Of course one can speak of catabolism only where there is, or once was metabolism. This expression is borrowed from chemistry, where the molecule passing through various processes, now breaks up and now again is built up anew. In biology (physiology, pathology) the expression "Dissolution" has not yet met with definite acceptance; but in my opinion it is not a poor choice, and might with advantage find application even in the discussion of the sphere of emotion and morality, where—in the "somatic" components—chemistry certainly plays a prominent rôle.

The terms "construction" and "destruction" may be used in various senses in biology: for instance, in that of biochemistry and biophysics, but also in the morphological sense. And one must have regard in both of these senses for the fact that in life processes there is a constant construction and destruction (as for instance in digestion and assimilation), but also too a construction and destruction of more stable values (furnished through long periods), which physiologically split up into subordinated factors, and these on the other hand can also be enriched by higher ones. Finally we come to a destruction where there is often no reconstruction: in acute and chronic, primary and secondary degeneration in the nervous system (necrosis or atrophy of fibers and of neuron elements). Through such destruction the nervous function becomes weaker; is perhaps mutilated and finally suspended. Indeed we can suppose an analogous "destruction" in the psychic sphere; that is to say: the acute or successively progressive loss of orientation, intellectual or moral aberration, and finally dementia.

We might expect that the more complex the construction of a function is, the more layers it is composed of, the more various the forms of the destruction and the greater the number of fragments and loosened foundation stones. The groundwork and foundation in the realm of emotion and morality are without doubt based on the primary instincts, that is, the instincts which have once created the functions most important for life, and have accompanied them without interruption; that is, the instincts important for life: air and food hunger. These indeed could not be further decomposed. What is decomposed in the realm of emotion are values which through culture and education are built up on fundamental instincts, through the mediation of countless ideas, being assimilated from these ideas. These are especially: greater regard for posterity, for the

family, for comrades (fatherland), then for the community ("the inalienable rights and duties of mankind"), and finally for the welfare of the whole cosmos, especially for man as such (humanity).

The tendencies just now sketched create the final goal of the germinal impulse towards perfection dwelling within every living creature, even within the human egg cell. The new emotional values built up during the phylogenetic and ontogenetic development, and in the closest relationship to the world of experience (a long, exceedingly delicately graduated register) hold their own against the primary emotions, and continually conflict with them. And the so-called psychic calm joy in life, contentment, happiness, represent a favorable balance with reference to "possession," in the distribution of the various forces here in question.

The primary emotions and impulses, as for instance, desire, love, hate, pain, aversion, the impelling will, as we have already pointed out, permit of no further destruction, at least biologically and psychologically. They constitute roots and stem of the whole emotional life, and as we have indicated must be ascribed even to the living protoplasm—to be sure, only germinally. Virtues and vices, on the contrary, the successively acquired egoistic and altruistic impulses, and the emotions derived from experience, that is, from stimulation of a corresponding latent state, which become manifest in the immediate present: compassion, enthusiasm, feeling of duty—to name only a few—can be decomposed, (perhaps only temporarily), that is, diminished, inhibited, repressed, annihilated, and indeed without harm to the accompanying ideas; and these impulses in connection with other relations be supplied with richer deposits and sprouts—that is refined; and both these processes take place in connection with numerous emotionally toned experiences, each such experience releasing in us a conflict with the primitive instinct and putting our "character" to test just as is the case through poisoning (alcohol and so forth), under influence of disease producing substances, and the like. Biochemically such a destruction perhaps may be considered as a competitive conflict between the autonomic and the sympathetic nervous systems (influence on the blood and in the cortex) on the one hand, and between the various "derivatives" from the activities of these two visceral systems, on the other. This is a conflict, the decision of which lies in part in the force and functional strength of the accompanying ideas earned through education (reason). The assumption just now expressed as regards the sympathetic and autonomic systems is supported by the antagonism of these two systems in the economy of everyday life. In the peripheral areas

of the two systems the antagonism reaches expression as is well known even pharmacodynamically, and furthermore through the results obtained by experiments with the secretions (Pavloff).

Emotions and morality possess, as we have seen, a well characterized, genetic construction in which we can distinguish a series of phases closely connected with the immediate problems of actual age. In a roughly schematic way we might present, perhaps, the following "biological scale" for the emotions and morality:

1. First phase: capacity to keep the most important functions running, as well as to compensate for "tensions" arising in this connection (gratification of sexual and food hunger).

2. Capacity to protect one's self from attacks in the separate bodily parts or organs, to nourish and care for the body. The hunger for food is appeased by the acquisition of food, which is connected with locomotion. In short, the capacity to maintain one's self within the habitual circle.

3. Striving for the affection of another, childhood love; later the first love for the opposite sex emerging timidly, longing for comradeship, joy through a symbol, through muscular exertion and games. Impulse to be active in this direction.

4. Desire for work, for development of one's own abilities. Need of knowledge, education for vocation, imitation of activities, of those in higher positions, development of character.

5. Increased security, avoidance of danger, defense against foreign unjustified encroachments, desire for conflict, unfolding of more mature sexual emotions, winning affection of the opposite sex by showing admirable personal characteristics, defense of honor, choice of a vocation, formation of an aim in life.

6. Care for the family and posterity. Education of children, establishment and settling down, greater provision for social duties undertaken.

7. Greater interest in the welfare of the community, active participation in social problems, fulfilling duties towards the community and in service of the public, love of country, and devotion to patriotic interests.

8. Constantly increasing development of the comprehension of humanitarian activities, appreciation of the rights of others, even outside the boundaries of the fatherland. More earnest religious, philosophical, humanistic tendencies and activities; active and passive activity in this direction.

9. Cultivation of a higher, altruistic nature, moments which de-

termine the noble character and disposition of man: self-sacrifice, a strict sense of justice, modesty, wise self-control, judicious acts of charity, and so on. The highest human ideals.

These levels of emotion and morality, up to the well established personal character, grouped in a roughly schematic way (related to one another in many ways, and without following one another in invariable succession) are formed as they occur in the everyday life of man. They are indicated partially, also, even if only in a rough way or in embryo, even in the higher animals living in groups or herds (in part indeed even in insects: for example, ants). At any rate certain "collisions and conflicts" between the various emotional values are present already in them. The latter are similar to those already mentioned, are organized, and demand discharge. In youth the instinct of self-preservation dominates, in the more mature individual the sexual impulse and the instinct of acquisition, in old age, social and religious impulses, and the preservation of what is acquired in life (frugality sometimes reaching avarice).

There now arises the question of a definite, degenerative dissolution of the emotions (produced perhaps by chronic intoxication, for instance alcoholism, or through sickness, for example progressive paralysis, dementia praecox, and so on); these emotions developed last in life and those depending on complicated education and discipline crumble away; and those characteristic of early youth (that is, emotions and impulses and appetites resisting harm of all kinds) now dominate the field. Frequently also the superfluity of "positive," healthy emotions (joy in life, and so forth) are injured, and the negative emotions (depression, bitterness, and so forth) win the upper hand; sometimes reaching the state of "chronic attitude of defense" against dangers that are mainly imaginary (persecutory ideas). In all chronic or progressive (infectious) diseases of the cortex (also in dementia praecox), there is an effort to thrust aside something standing in the way of the personal welfare of the individual, something "offending" to the emotional life, to make the attacks on the part of the outer world harmless, and so on, and this undermines every joy in life for the patient through a certain vicious circle (morose character).

Elementary self-preservation or "the chronic attitude of defense on the patient's part, against imaginary dangers"; this "latent war of independence" in one's own personal life constitutes from this point on the germ of the entire psychic life of the patient ("dissolution of the emotions"), while, on the other hand, the love for the

other sex, or for the profession, the regard for others and, finally and most completely, interests in all social activities, undergo radical decrease, or go under in the struggle with the primary instincts. Doubtless the emotional life may be attacked or decomposed in a relatively independent manner, that is, without harm worth mentioning to the true intellect, to orientation, or memory (criminality, moral idiocy; for instance in microcephalic cases). In this connection too, the conditions in many cases of chronic alcoholism are characteristic. Here, as is well known, the intellect, in so far as a certain sociability in the form of superficial conversation such as is carried on in saloons is concerned, is not noticeably weakened, nor are the sexual life, business, the every-day duties; on the other hand, there is displayed negligence towards family affairs, unreliability in business and in social relations, apathy towards higher interests; in general, forgetfulness of duties, inclination to lie, and so on, often in unmistakable manner. And, above all, strength of will and self-control are enfeebled. It is doubtless a question here of a relative or elective (sometimes only temporary) "dissolution" of higher ethical values, acquired slowly in a later period of life through education, and the discipline of daily duties, while the remaining traces of morality often assume infantile forms. The alcoholic, and even the sufferer from dementia praecox, doubtless in many directions of his emotional life regresses to a very early stage of childish development: the chief interests from this point on are related to the immediate present, to the immediate, personal (physical) comfort; that is, the instinct life strongly dominates. Care for the future is nearly extinguished, and the wishes take on an elementary character. The deterioration in cases of dementia praecox of this sort can go so far that only interests for the most vital bodily functions, and for (fragmentary) pictures from the remote past, are present. An attitude has been reached which coincides quite exactly with that of a small child, only that the drive towards perfection and broader development has been permanently lost.

Morality, especially the emotional life, in the psychoses, can sometimes be destroyed in manifold forms and with variations to any level. But this always takes place in the form of a "regression to a primitive grade of culture," or to levels which come to light in typical form in children and animals. And if it is a question of a degeneration of the individual, then it occurs under a continual decrease of the impulse towards higher goals, or perfection. The patient may remain, however, at any level of deteriorated morality (demoralization).

And what rôles do the nervous system and the inner secretions play here? Is it a question here only of biophysical, actual, and latent forms of excitement or of biochemical influences also? These both certainly enter into the question; the lion's share in the disintegration of the emotions, especially in the decrease of morality, must, however, be ascribed to the influence of the inner secretion, both active and latent, dependent upon the visceral and sympathetic nervous systems. In this connection, in relation to the latent emotional values one may recall the persisting energy of growth of the "fossil Egyptian grains of corn"! Every strong emotion and emotional value may correspond to a special chemical composition of the secretions of a special gland (though usually under similar conditions); the effect of this composition taking the character of a means of preserving an equilibrium in reference to other glands (antagonism between the adrenals, hypophysis, thyroid, and so on?). Probably the various inner secretions serving for the immediate preservation and for the continuance of the race, which are driven to the ganglia in question by the primitive mnemes, are those which flow most richly.

Many chemical materials work as a direct ferment, and specifically on the visceral and cortical nervous apparatus in question. Thus possibly there might be "defensive ferments" for the emotional life, just as for foreign intruders in the blood. But all these are of course only hypotheses.

In chronic alcoholic poisoning and in dementia praecox it is possible that only the nerve cell complexes serving the inner secretions, possibly only the cortical representatives of these (anatomical basis for the affect), are inadequate to the many sided demands made on them, and perhaps only the chemical combinations for the emotions immediately entering into action are produced, and perhaps also certain chemical by-products are set free which cannot be rendered non-intoxicating or can only be partly rendered harmless (lipoids in the blood?).

In alcoholism it is a question usually of a poisoning which attacks a great number of organs and cellular groups. Everywhere the most complicated compositions are the easiest to decompose.

One can even speak of a decomposition of emotions and morality in healthy and well-dispositioned men. Here it is usually a question of briefly enduring, reactive conditions following release of violent emotion, of suggestive effects, or of life conditions and relations depressive to the mood, or bringing bitterness and despair. Less important forms of emotion win the upper hand easily. One must

consider these forms as temporary "reactions" and there is usually a quick spontaneous return to normal through removal of the irritating moments as well as through moral counter-currents.

Indeed, the most simple release of a crude passion in an individual otherwise normal is regarded as an acute decomposition of morality under the influence of hypersecretion of the glands (adrenals?). It is inaugurated through a biological struggle over the interests most important at the moment for the individual's life, that is, for his immediate personal life or for the immediate future—interests which only too easily win the victim over the higher, humanitarian, altruistic interests, directed to the most remote future. The individual pays for momentary gratification of his crude instincts, if, for example, he permits an inconsiderate reaction in the interests of self-preservation (deed of violence) by another feeling of opposite nature, as the balance is restored; in the form of new emotions of different nature, that is, with remorse, feeling of guilt, which are suppressed into an existence in the latent state and the well-being of the carrier may be "poisoned" for a long time.

In these phenomena of dissolution, exhibited within certain limits by healthy people, phenomena whose foundation is to be sought usually in acute insults, menaces of various kinds (from those against the most primitive life interests to those against the most noble moral good), the object of the conflict is—as we have mentioned before—self-preservation, well-being and thriving condition of the family and members of the family or of home, and finally the ideal values of the world and the outlook for the future of all these. We have here the abandonment of the individual primitive instincts in favor of future interests, and especially in favor of the interests of society or humanity, or *vice versa*. These values are regarded as of greater or less importance, either consciously or unconsciously, according to the insight at a given moment, the emerging affects, or the education and culture. The balances or resultant consequences (advantageous or injurious for the individual) become the measure of the character of the personality. It is self-evident that subjective causality plays an extremely important rôle in these conflicts of the soul, which, for the most part, take place in the unconscious; also that the resolution into simpler elements (victory of inferior elements) may be progressive, and that the whole mentality of the individual may be thus unfavorably distorted (disintegration of the character). I will not here discuss the specific forms which this decomposition may take. There is no doubt that it may be produced through the influence of the inner secretions, as well as in other ways (that is, through the release of primitive instincts; by regression to earlier developmental

stages of emotion and morality; or by an undue stability of elementary forms of emotion, with the subjective causality that goes with these conditions). It will be sufficient in this connection to merely call attention to the different forms of fear (despair), attacks of rage, the assumption of the attitude of self-defense without cause therefor, inclusive of the use of cunning, etc.; to the "retrospective transformation of the wish into realization" in the form of lies; to the joy in harming others; and further, to the wild outbreaks of normal and perverse sexuality and the rage to destroy—two tendencies at times occurring simultaneously—etc.

These phenomena, which are transient in nature, are not brought about by pathological processes, strictly so called, nor by degeneration of the nervous system, but are, as it were, physiological dissolutions, and are connected with the emotional sphere. They may affect whole peoples in stirring political periods, in struggles for religious values and the rights of man, in telluric catastrophies—varying slightly with the special tendencies, the degree of education and of suggestibility, of the individuals composing these peoples. In similar manner they make their appearance in the daily life of the individual, in the passionate struggle to protect vital interests when threatened, especially sexual interests. In all these cases, in my opinion, there must be assumed a poisoning or flooding of the blood with certain inner secretions (a release of the herd instinct, for example, taking place not infrequently through suggestion), with a transformation of the stimulation into a latency stage. The fact that under some conditions the highest ethical values are developed concurrently with these explosions of passion (as contrast formations) alters nothing in their nature, and they must be regarded as phenomena of acute moral decomposition, the ethical values being ephemeral compulsive explosions of "pseudoaltruistic" emotion which have little in common with real morality proceeding according to the laws of calm reason.

At all events, in these "emotional conflagrations" violent outbreaks of instinct occur only too often, and these invariably correspond to those developmental phases which belong to the earlier evolutionary stages of morality.

FINAL OBSERVATIONS

(The Struggle to Preserve Psychic Equilibrium and to Attain Emotional Contentment)

If one looks beyond the subjective aspect, emotion is a physiologico-biological process (a tension seeking release), and, among other ways, manifests itself in the details of unconscious wishes,

urges, and voluntary trends in the living protoplasm, or the collective cell hierarchies, or, in higher animals, in the central nervous system (cortex). There sets in a constant blind striving toward some goal useful for the individual in some direction or other. Ethical perfection, the subjective reflection of this tendency, in which the "beautiful moment" of Goethe plays an important rôle, is attained only by successive stages, and never to the satisfaction of the individual. Full security of position and life, complete peace and perfect contentment are only possible for brief moments—moments not rarely attained by great effort. The prospects for the future which we strive to endow with the same emotions of satisfaction are in reality only phantoms. Man strives untiringly but vainly to realize this dream, to which he gives the name of permanent happiness. But at each advance in these successive stages of "happiness in the immediate present" new difficulties arise. Only in conditions of intoxication (as after taking alcohol) are all the shadows removed from the prospect of the future—and then only temporarily. This condition of intoxication is the so-called illusion which may be produced in man by a transient favorable constellation of stimulations in the nervous system. We can easily understand the causes of the sense of inadequacy (disillusionment) if we cast a fleeting glance on the not always modest demands of the various organs and the latency stages constantly arising and extending farther therefrom, as well as on the numerous different goals striven for with equal degrees of longing: from the feeling of general bodily comfort, air to breathe and food to eat, on to the highest ethical considerations, the enjoyment of art, etc.

A typical example of these conditions is that the ceaseless travail and effort of the cultured man to attain a permanent and entirely satisfactory position in life—to conquer a situation of contentment and the prospect of a peaceful future, is followed by a sudden disillusionment—often at the very moment when the long-dreamed-of plans have been brought to a happy realization. The love and honor of those belonging to him; the recognition of his merits and the high esteem of his fellow men, which he has earned by the services he has rendered; the assurance of material ease in the "future" for himself and for those dear to him, which at the end of his life he has attained and which also to a certain extent casts a pleasant reflection on his past—all these benefits lose their luster and their power to satisfy as soon as the endeavors and travail through which he has attained them come to a standstill. The honored statesman whose

counsel is sought by an entire people, if he is not senile when he retires from active life, is in just as problematic a situation, as far as actual happiness is concerned, as the restless industrialist, or the peasant who, having been throughout his entire life engaged in successful enterprises, seeks, in advanced years, to pass the remaining years of his life in comfortable idleness. All these persons find only a very small part of the satisfaction they anticipated from a quiet life, and they usually arrive at the conclusion that the real pleasures of a passive and monotonous existence are incomparably less than those of a life of endeavor, even though the latter be filled with cares and anxieties. These are all facts which have long been known, and may be expressed as follows: The goal which we strive unceasingly to reach as the reward of our endeavors is illusory. It is not the final arrival at this goal which brings us true happiness—but the toil and striving toward that goal.

Enough! The joy of life, the peace of the soul, happiness, do not reside in us as an independent quality, as it might seem to us. It is only attained by the rational use of our powers, by the wise satisfaction of the instincts and emotions, by the observation of the requirements of morality. The benefits which are thus arrived at must, however, be conquered anew every day and every hour. In a last analysis they are based on a physiological and biological equilibrium of the nervous structures and of the proper inner secretions. The acute access of pleasure, so-called joy, is essentially fleeting in nature and is a relatively short phase in the development of the positive affect (for example, in the form of the honorable sexual life). "Joy" in its raptures and its transports carries within itself the seeds of death, *i.e.*, the germ of its negative or opposite complementary affect, and often vanishes even before its climax is reached. Only by repeatedly surmounting the external and internal obstacles, through the proper cultivation of the emotional life and the character, supported by the constantly active urge toward perfection, can an adult cultured person attain that which seems to be naturally granted to animals and children (who seem to live in the immediate present), namely, harmless pleasure in life. This involves biologically (as does inner contentment) the favorable sequel of a wonderfully organized struggle in the economy of our cell hierarchy, especially in the central nervous system, of a struggle for the preservation of the equilibrium of the soul, in which the following factors, the value of which we have already sufficiently emphasized, are included: bodily well-being, sexual interests, the most restricted and the wider social interests, and the personal relations to God and humanity.

In this ceaseless struggle, embracing so many factors, into the details of which consciousness can never penetrate, the separate cells of the various sorts of tissue take part in obedience to the mnemes, seeking to preserve their individual and collective existence. The real protective agent of our bodily well-being and our highest moral interests, *i.e.*, the central nervous system (especially the cortex), is the most important element in this struggle (though it is not the sole important one). Here are made the decisions as to what is best for each organ, for each phase of life, for each moral value, and above all what is most expedient generally, that is to say, when and how the interest of the immediate present should be sacrificed to future and higher interests.

In healthy and well brought up persons, in the sphere of the central nervous system, just as in the motor sphere, the inhibitive apparatus with reciprocal relation to the powerful stimulative apparatus is well developed and organized. These two apparatuses are always in condition to influence and complement each other, provided the equilibrium in one direction or the other has not been disturbed by poisons, pathological processes, disastrous emotional experiences, catastrophes, etc. It is also indisputable that what we call the purpose of life and morality is the result of a conflict between emotional factors acting in contrary directions (the worst vices and the highest virtues are found side by side "in our breasts"). The physiological basis of these elements is to be sought, not only in the collective activities of the structures of the nervous system, or in the cortex, *i.e.*, in the so-called "world of ideas," but also in the inner secretions which work in, and with, these structures, that is, they are to be sought also in the biochemical factors of the unconscious volition and the mysterious primitive mnemes.

And Goethe, when he touches on this problem from a poetical or philosophical point of view, certainly hits the nail on the head when, in the second part of *Faust*, he makes the dying Faust say:

Yea, to this thought I cling, with virtue rife,

Wisdom's last fruit, profoundly true:

Freedom alone he earns as well as life,

Who day by day must conquer them anew,

So girt by danger, childhood bravely here,

Youth, manhood, age, shall dwell from year to year.

—(From Swanwick's translation of *Faust*.)

(Conclusion)

SOCIETY PROCEEDINGS

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

REGULAR MEETING, NOVEMBER 15, 1923, AT THE BOSTON
PSYCHOPATHIC HOSPITAL

F. K. HALLOCK, M.D., PRESIDENT, IN THE CHAIR

A CASE OF NARCOLEPSY

DR. KARL M. BOWMAN

A year ago at the meeting of this Society I presented a case of narcolepsy of nine years' duration in an unmarried girl of twenty-two. The attacks started at the age of thirteen, following an attack of chorea. The physical examination of the patient on admission was essentially negative. There was an abscess of the right lower central incisor, and a right internal strabismus. Other neurological and serological findings were negative. The spinal fluid sugar was 65 millograms. The x-ray examination showed the sella to be of normal size with possible slight erosion of the anterior face of the dorsum. The anterior clinoids were very long and almost met the posterior clinoids. The last basal metabolism in this case was minus 18 per cent and there was a definite tendency towards a flat or reverse type of blood sugar curve. On the basis of these findings the possibility of an endocrine disorder, particularly of the pituitary, as the basis of the narcoleptic attacks was considered and since June, 1922, the patient has been under organotherapy. Principally various pituitary and thyroid preparations have been tried. Pituitrin was given intranasally and subcutaneously with no particular effect. Posterior pituitary likewise showed no effect. Thyroxin intravenously raised the basal metabolism but failed to alter the blood sugar curve, and produced very slight if any improvement. Anterior pituitary seemed to produce some definite improvement although slight. Whole pituitary produced definite and marked improvement. Various preparations of thyroid, whole pituitary and ovarian substances have been tried and at the present time the patient is on a mixture of thyroid grains one-third, whole pituitary grains two, three times a day. There is marked improvement in her condition, and in order to test the effect of medication it has been discontinued for brief intervals at which time the narcolepsy invariably increases. Furthermore, the patient has, at times, neglected to take the medicine for a day or two and it has been noted that her symptoms became more pronounced at such times. The condition has not been cured by endocrine therapy but it seems to be demonstrated that the endocrine therapy has a definite beneficial effect in improving the narcolepsy.

A PSYCHOSIS DIFFICULT OF DIAGNOSIS

DR. WM. HERMAN

A woman of forty-eight, married twenty-eight years, a devout Baptist, was brought to the hospital because she had heard the voice of God and the Holy Spirit telling her to cross a marsh to save an outgoing ship; who had also heard God's voice telling her to pluck a perfect bunch of grapes from vines already picked clean. She later stated that when she found a few imperfect grapes she was led to think of people who were imperfect in their religious faith.

At this time she seemed to have unusual physical agility; whereas, a few days before, she had felt a good deal of slowing in her thinking and general working activity.

In going over her organically nothing positive had been found; her basal metabolism was negative; partial hysterectomy fourteen years ago; no flow since. She had three sisters with depression and ideas of reference at time of menopause, between forty and fifty. She was one of a family of seven. Her early life and social background is of greater importance. She was born in a small town in Vermont. Her father died when she was young, and the mother went out to work. The children were placed in different families. The patient did housework and waited on tables. At fourteen she had a premarital sex experience; she felt she could not marry a man with whom she had sinned. She came to Boston when nineteen. She married a Tufts graduate secretly; the marriage came out after six months. She was under a constant strain lest her behavior should not be suitable for the group into which her husband took her. To the one child, a girl, she wished to give all the things she herself lacked. She was very ambitious for her and sent her to Holyoke; but the girl was quickly withdrawn because the atmosphere seemed somewhat worldly. She was then sent to Boston University. About a year ago, the girl took a course in evolution, and once on coming home told the mother the Bible was a myth. The husband was not religious, and the patient's greatest conflict was that she would be called at Christ's second coming and her husband and daughter would be left. Up to a year ago she was under the constant guidance of a pastor, who knew all about her life. He represented "God on earth" to her and "was more than a father to her." He died a year ago and she now has no pastor she cares for. This culmination of strains, her feeling of social inferiority, worry over her daughter's salvation, and the loss of her spiritual guide, coming on at the time of the additional strain of the involution period was just a little more than she could bear. It is very hard clearly to place this case. She also had an unusual ability to achieve physical things, such as climbing the ladder and crossing the marsh without fatigue. Prior to the episode of the grapes, she suggested a good deal of thinking difficulty. There is surely the admixture of affect. The voice tells her to reach

this perfect bunch of grapes and she makes arrangements to perform this unusual thing. There seems to me here a certain amount of primitive symbolic thinking, and her own comparison of grapes to people of imperfect faith is easily given. Her treatment has been rather vicarious. She accepts very little in the way of suggestion, but it seemed to me that we could replace some of the props she has had. The daughter has been asked not to talk about evolution and not to criticize her mother's English, which she has been in the habit of doing. The local clergyman has been shown that he does not represent God on earth to her as the other man did and advised to try. The patient is in excellent contact and gives a clear retrospective account of herself as you have seen. She has fair insight and with the affective admixture and the improved understanding of the family, I believe she will adjust satisfactorily as long as home conditions are favorable.

DISCUSSION

Dr. C. M. Campbell: It is certain that the contents of her ideas are closely related to the problems of her life. As to how far it is possible to explain the complete breakdown on the basis of the problems thus revealed, there might be a difference of opinion; her physical equipment may have been reduced to a lower level. The episode occurred in a perfectly clear setting; the patient knew exactly where she was. If she had been confused and disoriented, the condition would have been a delirium with a strong religious coloring. We may find that as the emotion subsides she will get a certain insight into her past experiences. I think the prognosis is more favorable because the onset was quite acute.

Dr. D. Gregg: Why did you eliminate menopause when a partial hysterectomy had been done?

Dr. Herman: Because there had been no flow and no important symptoms; and I should have added, the absence of any sex desire following this operation. Her sex life practically ceased a few months after the operation.

A CASE OF MORBID FEAR

DR. C. MACFIE CAMPBELL

I wish to demonstrate a case of morbid fear which had lasted for one year. The patient claimed that he had been held up one night, and he gave peculiar details of this episode, for which there was no corroboration, and which probably was of the nature of a fantasy. He claimed since then that he was afraid of the Black Hand, and finally he had refused to leave his home. The one association he gave for the Black Hand was a reference to a schoolmate whose father had been killed by an Italian society. The relationship of the mechanism of the case to the obsessive mechanism on the one hand and the schizophrenic type on the other is of special interest.

ON AFFECTIVE REACTION-TIMES

DR. F. L. WELLS

Experiments were described in which visual and olfactory stimuli having various affective values were presented and the subject requested to indicate as soon as practicable the pleasantness or unpleasantness of the affect. The time required to do this was measured. In general the time of this process is of the order of eight-tenths of a second. A moving-picture technique was illustrated for measuring the time of involuntary emotional responses, such as facial grimacing. These times appeared considerably shorter than the voluntary reactions measured, of the order of one-quarter of a second.

DISCUSSION

Dr. H. C. Solomon: The facial grimaces might be thalamic reflexes, independent of actual emotional states.

Dr. Wells: This is the fact, which necessitates controls of an introspective nature, such as have been described.

THE SPINAL FLUID SUGAR

DR. BERNARD J. ALPERS

In this paper are presented the sugar estimations in 421 cases of mental and nervous disease together with some observations on apparently normal individuals. The sugar estimations in the spinal fluid were done in every case by a modification of the Benedict-Osterberg method for the quantitative determination of the sugar in the urine, modified by us for use in spinal fluid work. Each determination was compared with that of another worker who used the method of Folin. The results by these two methods varied only 2-5 mgs. per 100 c.c. of spinal fluid.

In our series thirty-three cases were chosen as apparently normal in that no physical defects could be found in the somatic system. In this group the spinal fluid sugar varied between 53 and 84 mgs. per 100 c.c. Most of the determinations fell between 53 and 68 mgs. per 100 c.c.

Determinations were made on 35 cases of epidemic encephalitis, the average figure being 82 mgs. per 100 c.c. The mean was found to be 84.5 mgs. per 100 c.c. Our opinion is that the test is of distinct diagnostic value, but is not pathognomonic. Other conditions may give spinal fluid sugars fully as high as epidemic encephalitis.

Twenty-five cases of untreated general paresis showed no increase in spinal fluid sugar, the average being 65 mgs. per 100 c.c. Several cases of untreated paresis showed sugar determinations as high as one obtains in epidemic encephalitis.

The sugar in treated cases of general paresis is not increased but is lower than that of untreated paresis, as shown by determinations in 163 cases.

Twenty-one cases of dementia precox showed an average spinal

fluid sugar of 80.1 mgs. per 100 c.c. One case gave a sugar reading of 103 mgs. and another 123 mgs. per 100 c.c. Our conclusion is that here is another condition which may give a spinal fluid sugar as high as in epidemic encephalitis though not as uniformly.

* We found the sugar in manic-depressive insanity to be normal. Two cases of diabetes mellitus gave sugar determinations of 123 and 189 mgs. per 100 c.c.

Numerous miscellaneous conditions are presented, many of which show a greatly increased sugar content in the spinal fluid.

USE OF TRYPARSAMIDE AT THE PSYCHOPATHIC HOSPITAL

DR. H. C. SOLOMON

Tryparsamide is a drug of the arsenic series which was compounded at the Rockefeller Institute in 1916 by Jacobs and Heidelberger coöperating with Brown and Pearce in their work with experimental syphilis. This drug, according to the reports of Brown and Pearce has a very marked affinity for the nervous system, and they find that in experimental trypanosomiasis it has quite remarkable curative powers. Similarly its effect on African sleeping sickness appears to be most satisfactory. Loevenhart, Lorenz, Blackwenn, and Hodges reported the results of their experience with this drug in neurosyphilis, finding it to give unusually good results. The chief difficulty in the use of the drug is its tendency to produce amblyopia. As a rule, this amblyopia is fleeting, but occasionally leads to permanent impairment of vision.

In June of this year, the Rockefeller Institute released a quantity of the drug to Dr. Ayer at the Massachusetts General Hospital, and to us at the Psychopathic Hospital. We have been using this drug since that time, and have had personal experience with about 70 cases of all types of neurosyphilis.

The object of this report is to call attention to the drug and the work that is being done with it, rather than to talk of results. We feel that our experience has been too limited to allow us to make any definite statement. However, it is interesting to note that the drug does something in cases of neurosyphilis that no other drug in our experience has done; that is, in every case of our series, without exception, ten injections of this drug has caused the cell count in the spinal fluid to become negative. In this series there were a number of cases that had been under treatment for a long period of time with other medicaments, despite which there was a pleocytosis, but this responded in every instance to the use of tryparsamide. Another striking feature is that nearly all patients develop a great sense of well-being when the drug is used and many gain weight.

Without attempting any further discussion of the results, we would merely summarize by stating that the drug is one that caused us, as the result of our experience, to have great interest in the continuation of its use of the drug.

DISCUSSION

Dr. J. B. Ayer: We have not treated so many patients at the Massachusetts General; we have had about twenty. We have tried to select particularly patients who have done rather poorly under other forms of treatment, thinking those a better test for the drug. Thus far we have seen nothing that could be called a brilliant result; but we have been using it only since June and on a small number of patients. We have not seen any paretics become normal or anywhere near normal. The clinical picture has not offered very much more.

As to the ease of giving the medicine, we agree with Dr. Solomon. It is easy and gives no reaction in any case. We have had two patients who have shown eye symptoms. One of these however would have developed symptoms anyway. The other I have treated since 1916 and suspected him to be slightly paretic. He has had slight symptoms all these years, and when under treatment with tryparsamide, optic atrophy developed for the first time (this is the first eye trouble we have seen in him), and it seems a little as if the medicine might have something to do with it. The other patient with one eye couldn't see ordinary print. The next day I had his eyes examined by an ophthalmologist, and after treatment he could see better than he ordinarily could. That was a transient amblyopia. Was it because of the tryparsamide, or was it disturbed circulation? I don't know. Either of these cases may be associated with the medicine. Again, I don't think we ought to judge it yet; it is too early, but of course having read the reports of Lorenz, it seems as though we ought to get something better than we have in the five months we have used it, so with a new supply of medicine we are going to continue with it, for it seems to be a safe procedure.

A Physician: I would like to ask whether the work done by the Public Health Service did not show a higher percentage than the Lorenz series? And how many treatments have been given?

Dr. Solomon: Work by the Public Health Service on amblyopia is entirely new to me. As to the length of treatment, Lorenz says it should be taken as long as is necessary if it takes years. Moore and Keidel varied the technique of Lorenz by eliminating the rest period; sometimes 18, 20, or 25 injections were needed before any change was noticeable.

FURTHER STUDIES IN BASAL METABOLISM

DR. KARL M. BOWMAN

AND

DR. G. PHILLIP GRABFIELD, M.D.

During the past two years over four hundred determinations of the basal metabolism have been done on hospital cases. The result of the first 50 cases was published in the *Archives of Neurology and Psychiatry*, March, 1923. At that time we called attention to the fact that low basal metabolisms were very common and that very few high readings were found. The result of further study has

been to confirm the previous findings. We would again call attention to the fact that the majority of errors cause an increase in the metabolic rate and that to obtain the true basal metabolism all authorities insist that the patient must be in complete physical and mental rest. Therefore, it seems that a number of our findings are probably too high.

The results of our findings may be briefly summed up as follows:

Seven cases in which we found no evidence of mental disease or defect were all within the normal limits of plus or minus 10 per cent and this affords us a good control for our other cases.

Further study in the organic psychosis did not confirm the tendency towards low findings reported in our previous article. A number of definitely increased rates were found in such conditions as general paralysis, lethargic encephalitis, etc. There was, however, marked variability in the organic psychosis, some being high and others low.

In schizophrenia, there was found a definite tendency towards low readings, over one-third of the cases being less than minus 10 per cent. This confirms our previous observations.

In the affective psychoses, the basal metabolism was usually within normal limits, the excitements were mostly minus readings and the depressions mostly plus readings.

Cases of mental deficiency showed a slight tendency towards increased readings but were mostly normal. This is of interest because of the common feeling that hypothyroidism may be a factor in mental deficiency.

In epilepsy, there was a definite tendency towards a low basal metabolism, over half of the cases showing readings below minus 10 per cent. In the cases of psychoneurosis and psychopathic personality, the basal metabolism was usually within normal limits.

DISCUSSION

Dr. O. J. Raeder: I was interested to hear Dr. Bowman's results in the cases of feeble-mindedness. I am engaged in that work with Dr. Fernald at Waverley, and we have had a number of cases under treatment. Unfortunately we haven't anything to add on basal metabolism, but in a general way we have noticed that in a number of cases there has been a great deal of improvement, both mental and physical, under treatment with thyroid and iodine, and in others with pituitary. I would like to ask Dr. Bowman if there was any one particular type of feeble-mindedness studied in his series.

Dr. Bowman: I cannot say offhand, but most of these cases I think were adults or persons over fifteen years of age, and there were no definite cases, as I remember at the moment, that were regarded as cretins or mongolian idiots. There were no particular physical findings about most of these cases; we had a number which seemed to be rather definite endocrine disorders, exophthalmic goitre, etc., but those are not included in the results reported.

CURRENT LITERATURE

I. VEGETATIVE NEUROLOGY.

2. ENDOCRINOPATHIES

Bailey and Bremer. EXPERIMENTAL DIABETES INSPIDUS. [Endocrinology, November 1921, III, No. 6.]

Even a small lesion of the parinfundibular region of the hypothalamus will occasion a polyuria in dogs which appears in the first two days after the lesion. According to the extent of the lesion the polyuria varies from a transient one lasting from six to eight days to an apparently permanent polyuria. In the persistent polyuria dogs other symptoms, *i.e.*, cachexia "hypophyseopriva," genital atrophy and adiposity were present. The polyuria has the chief characteristics of diabetes insipidus in man. Possibility of concentration when intake of fluids is restricted, when pituitary extract is injected subcutaneously or in the presence of fever, excessive polyuric reaction to the administration of chlorides, and absence of theobromin effect. The experimental diabetes insipidus the authors infer does not depend on a disturbance of a supposed nervous or vascular regulation of the kidney. It may be induced in animals whose kidneys have previously been denervated (an impossible thing to do, since the vegetative plexuses are even present in the blood vessels—Ed.) and when present persists with the same characteristics after denervation of the kidneys. Lesion of the tuber cinereum has produced in two dogs a cachexia "hypophyseopriva" with genital atrophy, and in two other dogs an insidiously developing adiposogenital dystrophy. The integrity of the pituitary was in each case verified histologically. Glycosuria was an inconstant result of the lesion and seemed probably to depend on the state of nutrition of the animal. The situation of this important nervous center and the minuteness of the lesion necessary to provoke characteristic symptoms probably explains the results of operations on the hypophysis in both young and adult animals. [As the authors do not follow up their experiments with serial sections of the brain, mid-brain, cord or sympathetics, and trace the degenerated pathways, as Lewy, Brugsch and Dresel have done, their work is incomplete and their hypotheses insecure. Ed.]

de Schweinitz, G. E. PITUITARY DISORDERS TREATED NONSURGICALLY. [Va. Med. Monthly, July 1921, XLVIII, No. 4. J. A. M. A.]

De Schweinitz points out that in stages of glandular insufficiency it appears to be a fact that the effectiveness of organotherapy is probably

enhanced by simultaneous administration of mercury (preferably by inunctions), and the gland extracts, even though the presence of syphilis is not demonstrable by the usual methods. It is probable that a combination of thyroid and pituitary gland extracts is more efficient than either of the extracts alone, and that this combination, associated with mercury is more effective than is an extract of one gland, even though given in conjunction with unguentum hydrargyrum. The value of radium in the treatment of pituitary body lesions has unquestionably been demonstrated in cases in which it has been applied both primarily and after operative procedures.

Bertolotti. RADIUM THERAPY OF ACROMEGALY. [Giorn. d. R. Acad. di Med. di Torino, March-June 1920.]

A clinical record of a man, aged thirty-five years, who came under observation with the signs of a rapid and progressive attack of acromegaly—namely, almost complete blindness, sexual impotence, profound adynamia, and advanced cachexia. A tumor of the hypophysis, the size of a small nut, compressing the optic chiasma showed on shadow. The patient was then subjected to radium treatment, amounting in all to 7,360 mg., distributed over eight sances during a course of four months. The irradiation was carried out exclusively over the skin of both temporal regions. The result of treatment is reported as follows: During the first stage, which lasted from February 8 to March 13, 1918, there was an aggravation of all the symptoms, especially as regards vision; in the second period (March 13 to July) there was slow but progressive improvement; and in the third period a clinical cure could be maintained.

Quick, D. RADIUM AND X-RAY OF TUMORS OF THE HYPOPHYSIS. [Arch. of Ophthalmol., May 1920.]

Discussing the varieties of pituitary tumors, attention is called to the high mortality and indifferent results from operative treatment. Kanavel estimates the operative mortality at from 10 per cent to 35 per cent. Cushing reports a mortality of 10 per cent in his own work. During the past few years X-rays and radium have been employed both as an aid to operative treatment and for the treatment of recurrences with promising results from many quarters. The author gives his experience of three cases. A woman of thirty-one had previously been operated upon by the nasal route. Five capillary tubes of radium emanation were embedded in the tumor through the nose. The headache and vertigo disappeared and the vision improved. The application was repeated eight months later. The second patient, a woman, aged thirty-seven, had had no previous operation. A resection of the nasal septum back to the sphenoid bone was done and the sphenoidal sinuses were opened, but the floor of the *sella* was not removed. A tube of radium emanation was introduced, packed with gauze and left in for twelve hours. Eight months later

radium was applied to both temporal regions. There was some improvement of the headaches and of the sight. The third patient had radium buried in the tumor after operation. He died from meningitis. The author concludes that X-ray and radium therapy should be tried before radical operation is carried out.

Geller. EXPERIMENTAL EXPOSURE OF THE PITUITARY TO X-RAYS. [Inaug. Dissert., Breslau, 1920.]

This is the record of an experimental research upon irradiation of the pituitary in nulliparous rabbits. The experimenter noted an apparent checking of growth in the hypophysis, especially in the anterior lobe and the pars interna media; histologically the small cells were most affected, especially perhaps the young forms of the large chromophile cells. The ovarian cells showed no characteristic alterations in appearance. The rate of growth of the animals appeared to be diminished. Similar treatment of a case of dystrophy adiposogenitalis was followed by increase of menstruation without alteration in the external habitus.

Roca, J. ON THE RELATIVE AMOUNTS OF DEPRESSOR AND BRONCHO-CONSTRICTOR SUBSTANCE OBTAINABLE FROM THE ANTERIOR AND POSTERIOR LOBES OF THE FRESH PITUITARY GLAND. [J. Pharmacol. and Exper. Therap., 1921, XVIII, 1.]

Abel and Nagayama have shown that the addition of mercuric chloride to a concentrated pituitary extract precipitates the pressor and oxytocic hormone; the depressor substance remains in solution. The author has used the nonprecipitable fraction to estimate the depressor substance. The glands were collected from slaughtered oxen, and the lobes separated at once. These extracts also contain bronchoconstricting properties. Extracts from the posterior lobe are seven or eight times more depressant than similar extracts of the anterior lobe. The bronchoconstricting action is also quoted. Chloroform extracts from both lobes in a dried state a substance possessing a histamine action. The posterior lobe yields more. The author believes it to be histamine itself. [Med. Sc.]

Timme, W. NONSURGICAL TREATMENT OF PITUITARY DISORDERS. [Archives of Ophthalmology, May 1920. J. A. M. A.]

In cases of underactivity, presumably of the anterior lobe, Timme advises feeding of the whole gland in doses of from one-half grain, as infrequently as once or twice a week, up to one or even two grains, three times daily, depending on the reaction of the patient to the dose, especially as regards the production of headache, excitability, insomnia, nausea and vomiting, and rapid fatigability. But in addition thereto, either feeding of the anterior lobe, or hypodermic injections of the anterior lobe extract, will often be found of value, especially in the Froelich syndrome with genital lack of development and in low bodily temperatures. Very small doses of thyroid gland, one-hundredth to

one-tenth grain, every day or on alternate days, will frequently enhance the effect of the pituitary administration. In cases of overactivity, treatment depends on the cause for overactivity. If this cannot be found and eliminated, then give sedatives such as luminal, one-half grain, once or twice a day, for a few days at a time; pilocarpin, one-tenth grain, or acetanilid, two grains, several times daily, as alternates. If disturbances in the genital sphere produce secondary pituitary activity, lutein or ovarian extract will be found of benefit. When the activity is secondary to a thyroidectomy, then small doses of thyroid gland will relieve the symptoms. In too early thymic involution, with pituitary overactivity, thymus gland, one to five grains, several times daily, is often effective. Occasionally, an inefficient chromaffin system will give rise to overactivity on the part of the pituitary, which may be offset by feeding whole gland suprarenal extract.

Dale, H. H., and Dudley, H. W. ON THE PITUITARY ACTIVE PRINCIPLES AND HISTAMINE. [J. Pharmacol. and Exper. Therap., 1921, XVIII, 27. Med. Sc.]

Abel and Nagayama believe that histamine is produced during the hydrolysis of the pituitary principles. This is denied by Dale and Dudley, who have subjected pituitary extract to hydrolysis and report the results of blood-pressure experiments and tests on the uterus of the guinea-pig. They compared the oxytocic and depressant action of the hydrolysate and of histamine itself. These do not correspond. Histamine, if present at all, occurs in such minute amounts that its chemical identification is impracticable.

Duffy, W. C. HYPOPHYSIAL DUCT TUMORS. [Annals of Surg., November 1920, LXXII, No. 5. J. A. M. A.]

Two cases of hypophysial duct tumors are recorded by Duffy. A previously healthy man, thirty-five years old, began rather abruptly to suffer with severe headaches, progressive diminution of vision, and loss of libido sexualis. Positive Wassermann tests resulted in antisyphilitic therapy. Later, the diagnosis of tumor in the hypophysial region was made by means of radiography. The visual fields showed a bitemporal hemianopsia. The exploratory craniotomy was complicated by unusual hemorrhage; however, the cyst presenting above the sella and between the optic nerves was evacuated, and the patient recovered from the immediate effects of the operation, but died twelve days later with symptoms indicating failure of the medullary centers (about one and one-half years after the onset of symptoms). At the necropsy of this slightly obese man a squamous epithelial intracystic papilloma was found presenting above the enlarged sella with remains of the anterior hypophysial lobe and traces of pars intermedia preserved in the basal sector of the cyst wall. Death apparently was caused by increased intracranial pressure (cerebral edema). The testes showed histologically a marked

atrophy. Thymus was retrogressive. Other glands of internal secretion showed no definite changes. Changes of subsidiary interest were found in the lung (bronchopneumonia, pulmonary infarcts) and appendix. A female child, eleven years old, who had suffered with headaches of increasing frequency for five years, progressive failure of vision for one year, and occasional projectile vomiting for eight months, was brought to the hospital because of increasing disability and the recent appearance of stupor. Instead of retardation of sexual characters, there was perhaps slight exaggeration of same. Radiography showed a suprasellar nodular shadow, due to calcification; partial destruction of posterior clinoids, and separation of the frontoparietal sutures suggesting a secondary hydrocephalus. At the exploratory craniotomy a suprasellar cyst containing 30 c.c. of fluid was evacuated and partly extirpated. Histologic examination of tissue from the wall of the cyst showed definite squamous epithelial cell derivatives presenting the picture of adamantinoma. Death apparently from cerebral edema. No necropsy.

Roth, H. CONTRIBUTION TO THE CASUISTICS OF THE TUMORS OF THE PITUITARY. [Beitr. z. path. Anat. u. z. allg. Path., 1920, LXVII, 309. Med. Sc.]

Description of a very large malignant adenoma of the anterior lobe of the pituitary body, observed in a man of twenty-nine years of age. The tumor had had a rapid development and had penetrated into the right lateral brain ventricle. Large secondary nodules were observed in the right occipital and temporal lobes. It appears from the author's investigations that tumors of the pituitary body of such a nature and size are of rather rare occurrence. [Da Fano.]

Sternberg, C. ON TRUE DWARFISM. [Beitr. z. path. Anat. u. z. allg. Path., 1920, LXVII, 275. Med. Sc.]

After reviewing the actual state of knowledge on the subject the author summarizes the life history of a case of "true" dwarfism on which complete histopathological investigations were carried out. The 92 cm. long dwarf was a male of seventeen years of age, dead in consequence of an old tuberculous spondylitis. Though the shortness of the body was in part due to the pathological condition of the vertebral column, all other characteristics were those of Hanseman's "*Nanosomia infantilis*." The body was of a normal size at birth, and his development had also been normal for the first eighteen months, when the subject fell from a chair. According to the information obtained from his parents he developed some time afterwards a very marked gibbosity and became a cripple. He went to school and was very intelligent. With the exception of the chronic tuberculosis of the spine and left femur the post-mortem examination showed the absence of any other pathological process. The limbs were very short and infantile; the bones extremely gracile with permanence of the epiphyseal lines of junction;

the genital and internal organs proportionately small. Beard, axillary, and pubic hair were absent. The endocrine glands were likewise exceptionally small but without any special regressive changes. The histological picture of the testicles, to which the author paid special attention, was that of an incomplete development or a true hypoplasia, without any trace of a secondary atrophy as sometimes observed in consequence of various alterations of the pituitary body. As a result of this investigation, and after a careful comparison with similar observations, both of his own and other authors, Sternberg comes to the conclusion that in the present instance the hypoplasia of the endocrine glands and the defective growth were coördinated manifestations of a general defect in the progressive development of the whole organism; in other words, a true case of Hastings Gilford's ateliosis. For this form of dwarfism, not connected with the deficient function or disease of any determined organ, Sternberg proposes the term "*Nanosomia hypoplastica*," reserving that of "*Nanosomia pituitaria*" for the cases due to an essential alteration of the hypophysis cerebri.

In accordance with these denominations Sternberg proposes the term "*Nanosomia thyreogenes* or *hypothyreotica*," to indicate a form of "true" dwarfism connected with the defective development of the thyroid body. Of this form Sternberg gives the following interesting instance: Dwarf 126 cm. long, dead at the age of twenty of an intercurrent pneumonia. His father had been an alcoholic and died at the age of forty-three of tuberculosis. The boy was of normal size at birth, but had learned to walk only at the age of six and to speak at that of eight. At ten he measured 105 cm. in length, which is approximately the size of a male child of six years of age; his intelligence was so little developed that he had to be sent first to a school and then to an asylum for feeble-minded children. In the following ten years he grew only another 21 cm. in length, but showed no intellectual development, so that he was considered an idiot. The secondary sexual characteristics never developed; the genital organs remained very small. At the post-mortem examination Sternberg found that the trunk and limbs were short but proportionate; the bones were gracile and of an infantile type. The internal organs and endocrine glands were likewise proportionately small. The histological investigation of the testicles showed that they had, at least up to a point, developed normally, and had then undergone a secondary atrophy characterized by a remarkable thickening and hyaline degeneration of the walls of the seminiferous tubules, many of which were completely obliterated. No changes were found in the endocrine glands with the exception of the thyroid body, the most conspicuous alteration of which was the almost complete absence of colloid. In addition, the vesicles of one of its lobes were lined by a high cylindrical epithelium, and were structurally similar to those of the thyroids of the new-born; only a few vesicles of the other lobe showed such a picture,

most of them being filled with cubical epithelial cells. This observation appears to justify the proposed separation from the *Nanosomia hypoplastica* and *pituitaria* of a *Nanosomia thyreogenes*, clinically characterized by defective though proportionate development of the whole organism, very slow but still steady body growth, partial permanence of the epiphyseal lines of junction, and chiefly by intellectual deficiency. It could be differentiated from the cretinic form of dwarfism because of the characteristic want of proportion of the latter. [Da Fano.]

Engelbach, W. CLASSIFICATION OF DISORDERS OF THE HYPOPHYSIS.
[Endocrinology, July-September, 1920.]

This author essays a generalization relative to pituitary disorders. Three divisions are made by him, dependent on whether one or both of the lobes are involved: (1) anterior lobe disorders, this lobe alone involved; (2) posterior lobe disorders, this lobe alone; (3) bilobar disorders, in which aberrant function of both lobes could be demonstrated. Each lobar division is subdivided into the activities prevailing, the anterior and posterior lobe disorders into the states of: (a) hypoactivity and (b) hyperactivity; the bilobar group into the state of (a) hypoactivity, (b) hyperactivity and (c) heteroactivity. The last comprises mixed cases, in which there were opposing secretory states of the individual lobes. A further division into: (1) pre-adolescent and (2) post-adolescent varieties, dependent on the age incidence of the abnormal secretory state is made. A final division is made of the age incidence, into: (a) a-neoplastic and (b) neoplastic varieties. Under the pre-adolescent hypoactivity of the anterior lobe is grouped Lorain-Levi type of pituitary insufficiency. In the post-adolescent hypopituitarism of the anterior lobe are arranged: female cases which have had some osseous changes indicative of early anterior lobe disorder, and who after maturity developed amenorrhea, metrorrhagia, or dysmenorrhea, dissociated from any local or general disease. Substitution treatment by anterior lobe extracts causes definite improvement. Under the hyperactivities of this lobe are described the clinical syndromes of gigantism and acromegaly.

Cori, K. PERIPHERAL ANTAGONISM BETWEEN VAGUS AND SYMPATHETIC, AND THE EFFECT OF THYROID ON THE HEART NERVES.
[Arch. f. exper. Path. u. Pharmacol., 1921, XCI, 130. Med. Sc.]

Every teacher of physiology has long known that during the summer months stimulation of the vagus rarely results in stoppage of the frog's heart. This is now shown to be due to the fact that sympathetic fibers run together with the vagus, and in summer this excitability is increased, while that of inhibitory fibers is depressed. The vagus effect can always be elicited in summer if the sympathetic terminations are first paralyzed with ergotamine. Or again, if a frog be taken in which vagus stimulation is followed by no inhibition, and a small dose of physostigmine be given, which by itself does not affect the heart-beat, stimulation of the

vagus will then bring about typical inhibition. In winter vagal inhibition can normally be obtained on electrical stimulation of the nerve, but if a dose of atropine be given, the sympathetic effect can be demonstrated. The change from the "winter" response to the "summer" response takes place at the breeding season. At that time it is common to find a frog which, while its own circulation is intact, gives an inhibitory response to vagus stimulation, but when its heart is perfused with Ringer shows a complete absence of inhibition. A frog which is selected at the time it is performing the sexual act always shows marked inhibition in response to vagal stimulation. Now thyroid extract has the property of abolishing an inhibitory response, that is to say, it has the property of increasing the sensitiveness of the sympathetic nerves and of diminishing that of the parasympathetic nerves. The author considers that there is a close relationship between the development of the thyroid in the summer months, and the changed response of the cardiac nerves.

Bittorf, A. PIGMENTATION IN ADDISON'S DISEASE. [Deutsches Arch. f. klin. Med., 1921, CXXXVI, 314. Med. Sc.]

By immersing strips of skin from cases of Addison's disease in dilute solutions of adrenalin darkening takes place. This is due to the oxidation of adrenalin, and the place of oxidation is in the epithelial cells of the skin. There is, therefore, an increase in oxydases in the pathological tissue.

Camus, J., et Roussy, G. THE SYNDROME OF OBESITY AND INFANTILISM IN RELATION TO DIABETES INSIPIDUS. [Compt. rend. Soc. de biol., 1921, LXXXV, 296. (Physiol. Abstr.)]

A dog, in which an experimental lesion at the base of the brain was produced at the end of 1919 has become enormously fat, while there are signs of testicular insufficiency. Its weight has increased from 15 to 26 kilos, and it suffers from diabetes insipidus, passing three to four liters of urine in twenty-four hours.

Cori, Gerty. EXPERIMENTAL INVESTIGATION OF A CASE OF CON-GENITAL MYXEDEMA. [Ztschr. f. d. ges. exper. Med., 1921, XXV, 150. Med. Sc.]

The heat-regulating mechanism of a myxedematous child of four years of age was compared with that of normal children of a like age. On placing in a cold bath the temperature of the former fell as much as 3° C. in the course of two hours and a half, while that of normal children was maintained, although both types of children showed external evidences of the cold bath. In a warm bath the myxedematous child did not perspire, and the skin remained white. It behaved as if narcotized. While the temperature of normal children rose, the temperature of the myxedematous subject was unaffected by external heat.

By the injection of foreign protein (3 c.cm. of cow's milk) there was a rise in temperature amounting to over 2° C. Pilocarpine and atropine exercise a more pronounced effect than with normal children. The child was treated with thyroid with evidently excellent effect (photographs) and the experiments repeated. In all cases, as shown graphically by charts, the metabolism approached that of normal children. It is noteworthy that in the myxedematous condition the child did not react to adrenalin with a glycosuria.

Uhlenhuth, E. INFLUENCE OF ANTERIOR LOBE OF PITUITARY ON GROWTH. [Jour. of Gen. Phys., January 1922, IV, No. 3.]

Amblystoma tigrinum when fed pituitary anterior lobe can reach a size far in excess of that of animals fed on earthworms and presumably also of that of liver fed animals. Liver as a food produces a rate of growth as high as that resulting from anterior lobe feeding, but maintains growth only, until the animals reach a definite size which is far below that of the animals fed on anterior lobe.

Lowe, F. M. DISTURBED PITUITARY FUNCTION ASSOCIATED WITH SPHENOIDAL SINUS ABSCESS. [Mo. St. Med. Asso. Jour., January 1922, XIX, No. 1.]

Sphenoidal sinus infection may be complicated by pituitary signs according to this observer. In the case reported by him of an unsuspected abscess involving the sphenoidal sinus was found. Dysfunction of the pituitary were the only symptoms observed.

Lopez Albo, W. PITUITARY IN ADIPOSIS DOLOROSA. [Archives de Neurobiologia, December 1920, I, No. 4.]

The author collects the Spanish literature. Less than ten cases of adiposis dolorosa have been published, he says, and adds one to the list. In his case pituitary, thyroid and ovarian treatment have done nothing. The variety of lesions encountered at necropsies shows various factors involved and the necessity for individualizing treatment. Pains in the temples and back of the orbit, congestion in the optic disk, and small sella turcica were the chief symptoms relative to the pituitary. Points of contact between this disease and circumscribed edema and symmetrical painful lipomatosis are discussed.

Schulmann and Desoutter. PITUITARY POLYURIA. [Rev. d. Méd., October 1920, XXXVII, No. 11.]

Diabetes insipidus is found to follow a number of different events. What causal relationship essentially is present is left open. These authors list among the direct causes traumatism, intoxications, infections and emotions, besides tumors. They state that 85 per cent of the cases on record were in individuals under twenty years of age, in a babe of two months and one of three years of age are noted. The pituitary

seems to be peculiarly unstable during childhood and at puberty, but diabetes insipidus has been encountered also in the elderly. Hereditary transmission of diabetes insipidus is recorded. A family in which the father had acromegaly and the son diabetes insipidus and glandular insufficiency is given by Marañón. All writers state that extract of the posterior lobe of the pituitary is the most effectual treatment, but it probably does not modify the prognosis. It reduces two distressing features, the thirst and the malaise which prevent sleep. Pituitary treatment may likewise act on other manifestations of secretory insufficiency, and it may sometimes be usefully reënforced with thyroid or suprarenal extract. They condemn any reliance on dieting or on drugs to act on the nerves since our knowledge of the coördinating mechanisms is still nil.

Seaman, E. C. IODIN IN SHEEP PITUITARY. [Jl. Biological Chem., August 1920. J. A. M. A.]

According to these observations there is no iodine present in sheep pituitary, an observation already demonstrated by Denis.

Bailey, Percival. STAINING GRANULES IN PITUITARY. [J. of Med. Research, June-September 1921, XLII, No. 4.]

Percival Bailey here records a staining method for the eosinophilic and basophilic granules in the same preparation. Such preparations seem to show that although the basophilic and eosinophilic cells arise from indistinguishable reserve cells they develop along diverging lines. The histological evidence is consistent with a view that the lipoid, colloid, hyalin and other changes occurring in the pituitary with age and after certain manipulations of the other glands of internal secretion are chiefly of degenerative nature.

Maranon and Solanilla. PITUITARY DWARF GROWTH. [Archivos Españoles de Pediatría, August 1921, V, No. 8.]

In this clinical paper the authors support a hypothesis that in all cases of true dwarfism a deficient functioning of the anterior lobe of the hypophysis cerebri is to be held accountable. In the case described a fifteen-year-old boy measured 1.24 m. and weighed 32 kg. Radiography showed the sella turcica abnormally small and flat. The case was classed by the authors as an adiposogenital syndrome plus pituitary dwarfism.

Gayler. DISTURBANCES OF GROWTH IN CHILDREN WITH DIABETES INSIPIDUS. [Monat. f. Kinderheilkunde, July 1921, XXI, No. 4.]

This patient of twelve years of age had been drinking five quarts of fluid daily for the past nine years. He sought to establish a relationship between the large water intake and increased nitrogen breakdown. He found such a relationship and also a marked increase in calcium loss. He does not go beyond the pituitary in his general reasoning.

Mintz. ACCESS TO PITUITARY THROUGH SPHENOIDAL SINUS. [Arch. f. Klin. Chir., January 27, 1922, CXIX, No. 1.]

This surgeon discusses this route. He turns back the nose and works back to the sella horizontally through the sphenoidal sinus. He illustrates the procedure in four cases which demonstrate the advantages of this method. The shape of the sella turcica is shown to vary greatly.

Amat, A. M. OCULAR MANIFESTATIONS OF DIABETES INSIPIDUS. [Siglo Medico, July 1921, LXVIII, No. 3529.]

In a man of twenty-nine with diabetes insipidus the symptoms indicated pressure on the optic chiasm. These symptoms had always been mitigated by reclining and aggravated by standing. They were much improved under pituitary treatment. The urine output dropped from 8 to 4 liters, but the atrophy of the optic nerves persisted unmodified.

Villa, L. DIABETES INSIPIDUS AND THE PITUITARY. [Policlinico, October 1921, 28.]

This clinical case report is of a severe diabetes insipidus in a man of twenty-six who had severe diarrhea from chronic enteritis and who died from debility. The pituitary was markedly affected and explained the diabetes insipidus. Chronic enteritis with diarrhea is not uncommon with diabetes insipidus. There is some causal connection but the author does not work it out.

Kay, M. B. HYPOPITUITARISM; FROELICH TYPE. [Endocrinology, May 1921, V, No. 3. J. A. M. A.]

Kay believes his case is one of the youngest cases of the Froelich type of hypopituitarism to be noted. The child was a full term baby; delivery was easy. It had always been breast fed and had suffered from no acute illness. Development was apparently in no way remarkable until the third month, at which time it was noticed that the weight began to increase rapidly. The mother also noticed that from this time on the mentality of the child was regressing; its stupidity becoming more and more pronounced. Sleep was almost continuous, and the waking moments were devoted almost entirely to feeding. The child did not sit up until the eighth month and at the ninth would not sit alone, and supported his head with difficulty. The feedings were taken quite well, and there was a mild constipation. A very marked polyuria was present. Treatment was started with thyroid extract, one-fourth grain, three times a day, whereupon the polyuria immediately disappeared, the protrusion of the tongue ceased, and the dribbling of saliva stopped. The thyroid treatment was gradually increased in amount, and very shortly pituitary (whole gland) was added, in increasing dosage, until at the present time, the child is taking three grains of thyroid extract and nine grains of pituitary extract daily. Almost immediately the somnolence decreased, the mental condition improved and the child now notices objects and

will reach for them. He can at this time (December 1920) stand by holding on to a chair. The weight remains constant.

Reverchon and Worms. LESIONS OF THE HYPOPHYSIS IN FRACTURES OF THE BASE OF THE SKULL. [Bull. et Mém. Soc. de Chir. de Paris, May 14, 1921.]

As the result of a motor accident this thirty-four-year-old man had a fracture of the base of the skull. There was bilateral paralysis of several cranial nerves (fifth, sixth, and seventh), but also symptoms of diabetes insipidus (polyuria and polydipsia), with intense anemia, asthenia, fall of blood pressure, and a mental state characterized by puerilism and apathy. The autopsy showed that the diabetes insipidus was due to extensive changes in the hypophysis, which was reduced to a small fibrous nodule in which no trace of normal tissue could be found on histological examination. This case suggests that the hypophysis is injured more frequently than is supposed in fractures of the base, either directly or by compression by a hemorrhagic effusion. This hypothesis is all the more likely as the hypophysis is a vascular organ very liable to hemorrhage. It is therefore advisable, in fractures of the base, to investigate not only the well known signs of this condition but also the symptoms of diabetes insipidus together with changes in the general condition and mentality. X-rays in such cases will be of value in showing lesions of the posterior wall of the sella turcica, which would justify the hypothesis of a pituitary lesion. If the clinical findings agreed with X-ray examination, pituitary opotherapy would be indicated.

Pérez. PITUITARY AND GESTATION. [Semana Médica, May 1921, XXVIII, No. 20.]

This author gives an historical summary of the development of knowledge relative to the physiology of the pituitary, and the effects of its removal in gravid and nongravid animals. He also details extensive animal experimental work, chiefly on dogs. He believes that these and other experiments have demonstrated that the pituitary hypertrophies during gestation give rise to exaggerations of its usual functions. In gravid animals removal of the pituitary is inevitably fatal in a few hours. Nongravid male and female dogs survived the hypophysectomy in one-third of the cases.

Gibson and Martin. PITUITARY EXTRACT AND HISTAMIN IN DIABETES INSIPIDUS. [Arch. Int. Med., March 15, 1921.]

Lumbar puncture, pituitary extract, histamin administration, the nitrogenous metabolism, the blood constituents, and the carbohydrate tolerance are here studied in their relation to a severe diabetes insipidus case complicated by syphilis. Lumbar puncture did not relieve the symptoms, the polyuria being rather increased on the day of the puncture, and on the following day. Pituitary extract given subcutaneously

increased for a time the concentration, and reduced the volume of the urine; histamin gave a similar though less effective result. On the day following the first pituitary extract injections, and on subsequent intervening nonpituitary days, a decrease in the polyuria with a relative increase in concentration was maintained. Desiccated whole pituitary substance, in four three-grain doses orally, produced a slight immediate effect. A lower nitrogen elimination resulted from the pituitary extract injections, with diminished ammonia and a somewhat increased uric acid output. Glycogenesis was not reduced, and there was hypoglycemia.

Aievoli. SURGERY OF THE PITUITARY BODY. [Riforma Med., Naples, February 12, 1921. J. A. M. A.]

This synthetic review of recent literature shows the progress in this line. In 160 operations on the pituitary that have been recently compiled, the mortality was about 50 per cent but is gradually decreasing. In the fatal cases meningitis was responsible for the deaths in 70 per cent. The operation serves mostly merely to relieve compression, but the results obtained to date justify intervention for such inevitably fatal lesions.

Rasmussen, A. T. THE HYPOPHYSIS CEREBRI OF THE WOODCHUCK (*MARMOTA MONAX*) WITH SPECIAL REFERENCE TO HIBERNATION AND INANITION. [Endocrinology, 1921, V, 33-66.]

Since 1915, when Cushing and Goetsch published their article "Hibernation and the Pituitary Body," in which histological changes in pars anterior suggesting a stage of hypofunction during winter-sleep of the American marmot were reported, many clinical papers have appeared in which it is assumed that the changes noted above are established facts and therefore strong support for the idea that in hypopituitarism one may expect a tendency to somnolence, lowered blood pressure, subnormal temperature, etc. Similar changes were reported earlier (1906) by Gemelli in the European marmot. The combined studies by Cushing and Goetsch and by Gemelli involved, however, only 29 animals and since Mann in 1916 could not corroborate these findings in the thirteen-lined ground squirrel (also a profoundly hibernating mammal), a thorough study was undertaken of the hypophysis of 32 woodchucks or American marmots (*Marmota monax*), the same species as was used by Cushing and Goetsch. In every case the hypophysis was fixed *in situ* immediately after death by the injection of the fixing fluid through the aorta. Complete serial sections were made and stained by several methods. Mallory's connective tissue stain, after corrosive sublimate fixation, gave the most differential tinctorial effect on the three types of cells in pars anterior. By careful volumetric methods (Hammar-Jackson) the volume of the whole organ and its various parts was determined for the three critical periods: just before hibernation, during the latter part of dormancy, and after becoming active in the spring,

both before feeding commenced and after feeding had been resumed for one to three weeks. The volume of the cytoplasm and of the nuclei of the cells in pars anterior and pars intermedia was similarly determined. A differential count of the three types of cells in pars anterior was also made.

It was found that there is apparently a sex difference. The male gland averaged 13 per cent larger than the female gland. This difference is equally shared by pars nervosa and pars glandularis; but, of the two components of the latter, pars intermedia was 50 per cent larger while pars anterior was only 10 per cent larger in the male.

Hibernation produced no changes in the weight or histological structure of the hypophysis when compared with the prehibernating gland, which should be the standard of comparison. The atrophic appearance of pars anterior (suggesting a stage of hypofunction), which have been reported by previous workers on the marmot, is not confirmed. On the contrary, the results are fully in agreement with those obtained by Mann from the ground squirrel. Immediately after waking up in the spring and during the onset of the rutting season, hypertrophy takes place in the hypophysis to the extent of 33 per cent. This increase is distributed proportionately among the three principal parts of the gland. The only conspicuous histological change accompanying this enlargement is a tripling of the relative number of basophiles in pars anterior and a distinct increase in their staining reaction.

It is believed that a failure to compare the hypophysis of the hibernating stage with that of the stage just before the onset of dormancy and the tendency to interpret the posthibernating conditions as the normal stage, are responsible for the idea that winter-sleep in marmots produces atrophic changes in the hypophysis. The return to the prehibernating condition evidently occurs late in the summer as is the case with the interstitial cells of the testis and ovary. There is a striking difference in the reaction of the woodchuck and of the albino rat (a nonhibernating species) to inanition. While inanition in the rat produces atrophic changes in the hypophysis (Jackson), starvation in the marmot even in the spring during several weeks of great activity and after three months deprivation of food during dormancy, is attended with no such characteristics but rather with hypertrophy of the whole organ and hyperplasia of some of its elements. There were an almost entire absence of intermediate forms between the three principal types of cells in pars anterior, even during the period of greatly increased activity of early spring. This militates against the theory that these three types of cells (chromophobes, basophiles, acidophiles) are only different functional stages of the same cell type, for were this theory true one would expect many intermediate stages especially during periods of great activity upon the part of the gland. [Author's abstract.]

Giannettasio, N. CRANIOTOMY FOR PITUITARY SYMPTOMS. [Riforma Med., February 19, 1921.]

The woman of thirty-three had been presenting for some time disturbances of the type of adiposogenital dystrophia, and other symptoms such as we are accustomed to see with tumor of the pituitary gland. A palliative decompressive craniotomy was followed by notable improvement, all the symptoms subsiding except the amenorrhea and hemianopsia; the latter was improved but still persists to some extent. Radiotherapy shows the sella turcica with better defined outlines now, evidently due to recalcification. [J. A. M. A.]

Vermeulen, H. A. HYPOPHYSIS TUMORS IN DOMESTIC ANIMALS. [Endocrinology, 1921, V, No. 2.]

Hypophysis tumors can arise in three different ways: (1) originating from rests of the hypophysis vesicle or from the epithelium of a canalis craniopharyngeus persistens, (2) originating from the ependym of the infundibular canal, (3) by hyperplasia of the glandular part. In veterinary pathology cases are known and described which demonstrate each of these ways. (1) In his dissertation (Bern, 1916) Wolff describes a hypophysial tumor of a horse, weighing 80 grams (normal weight about $2\frac{1}{2}$ grams), which had extended through a slit of the sphenoid into the sphenoidal sinus. In several domestic animals the nervous part of the pituitary body refers to the glandular part otherwise as in man; so in the horse it is wholly surrounded by the glandular part and in ruminants it is lying dorsally of the glandular part. In the "Bulletin Centrale de Médecine Vétérinaire, 1890," Mollereau describes a hypophysial tumor in a horse of nine years of the size of a hazelnut, which lies quite centrally; Falenta saw a like neoplasm in a cow, which had grown out in the center, from the nervous into the glandular part. (3) Author saw in two horses and in a hermaphroditic goat hyperplasia of the glandular part of the pituitary body. The tumors of the horses were surrounded by a strong fibrous capsule; on the cerebral side large hemorrhages were found. Except small cysts, more or less filled by a colloid substance and several bundles of connective tissue which crossed the tumors, only eosinophil cells could be observed. In the goat the hypophysis had five times the weight of the normal organ; enormous colloid-cysts occurred in the pars intermedia and also, but in less degree, in the hyperplastic glandular part. There was no occasion to examine the thyroid gland of this animal. [Author's abstract.]

Maranon, G. DIABETES INSIPIDUS A HYPOPITUITARY SYNDROME. [Endocrinology, March 1921. J. A. M. A.]

Marañón believes that the internal secretion of the posterior lobe of the hypophysis exercises, physiologically, a controlling action on the elimination of water through the renal filter and that, through the dis-

turbance of this controlling mechanism, diabetes insipidus is produced. In his opinion, the hypophysial oliguric hormone may, perhaps, act directly on the renal cell (either increasing its power to concentrate dissolved matter, or increasing its capacity to retain water), and partly by means of the nervous system, collaborating with the oliguric centers at the base of the encephalon as postulated by Pende. The modifications of these centers would be transmitted to the kidney, probably by way of the sympathetic, as some of the experiments seem to show. It is very probable, as Cushing and Biedl have suggested, that the hypophysial hormone ascends by the tuber cinereum to act on the proximal mesencephalic centers. Marañón and Rosique showed experimentally in one instance that the lesion producing a very intense diabetes insipidus was the fibrous scar which was produced around the shore which separated the hypophysis from the nervous tissue of the infundibular region. Marañón insists on the importance of the emotions in the pathogeny of this disease.

II. SENSORI-MOTOR NEUROLOGY.

2. SPINAL CORD.

McDonagh, J. E. R. EXPERIMENTS WITH CEREBROSPINAL FLUID. [Lancet, November 13, 1920. J. A. M. A.]

Shaking the cerebrospinal fluid with toluene, xylene or benzene the day after it is withdrawn, according to these experiments, affords a new criterion to separate degenerative lesions from other types. Another portion of the fluid may be treated with liquor formaldehydi before it is shaken with a hydrocarbon liquor. The various proteins merge into one another; hard and fast line between albumin and globulin, and lipoid-globulin are absent. The differences between them are physical. The complement-fixation test is regulated by the ions on the surface of the protein particles—their number and electric charge. The colloidal gold test depends upon the presence of lipoid-globulin particles, which have a positive surface electric charge. When carried out with distilled water this test will serve, says McDonagh, to distinguish a normal fluid from a fluid in patients with degenerative lesions. The latter will precipitate the colloidal gold in the first few tubes only. This may be confirmed with the fluid pipetted off from the hydrocarbon emulsion, because only those fluids from cases of widespread degeneration will precipitate colloidal gold. A diminution in the precipitation of colloidal gold in the first tube when the fluid has been treated with acetic acid indicates degenerative encephalitis. This may be confirmed by the slight precipitation which occurs in fluids which have been treated with ammonia and allowed to rest before being tested and which is only met with in cases of degenerative encephalitis. The lipoid-

globulin particles simulate parasites in certain ways and those found in the cerebrospinal fluid in degenerative cases are waste products which have reached the fluid by ultrafiltration through the choroid plexuses. Treating a normal fluid and serum with an acid makes the protein particles herein approach as near as possible to those normally met with in syphilis.

Arias, B. R. COAGULATION OF SPINAL FLUID. [Archivos de Neurobiologia, December 1920, I, No. 4.]

This clinical report shows the syndrome of Froin to have developed in the woman of fifty-two, two years after removal of the breast for cancer. Later a spastic paraplegia developed, and in testing the C. S. F. it coagulated spontaneously. Death from lung carcinoma soon followed.

Stern, L. CEREBROSPINAL FLUID. [Arch. Suisses de Neur. et Psych., Vol. VIII, 215.]

Stern reports a number of experimental tests of the fate in the central nervous system of various substances introduced into the blood of nephrectomized animals. Sodium bromid appeared regularly after 75 minutes in the cerebrospinal fluid and in the nervous substance in a lesser degree than in the fluid or the blood. Morphin and strychnin could be demonstrated after 20 minutes in the cerebrospinal fluid and in the nerve substance, in greater quantity in the latter than in the blood because of the great affinity on the part of the nerve tissue for these substances. Sodium salicylate, atropin, curare, etc., appeared after a time in the cerebrospinal fluid and the nerve substance, but adrenalin, eosin, uramin could not be demonstrated. This shows that there is a discriminating hematoencephalitic barrier through which only certain substances may pass although it permits the passage of all substances freely in the reverse direction from fluid to blood. This is important in medication as one may determine which substances must be injected directly into the fluid of the ventricle if the brain substance is to be reached. It was further proved that the substances found in the brain had arrived via the fluid and not directly from the blood. Stern presents a schematic outline of the circulation of the fluid as follows: blood > fluid of ventricle > nerve substance > fluid of subarachnoid space > blood. An article by von Monakow (*The Circulation of the Cerebrospinal Fluid*) on p. 233 of the same Journal confirms and extends the facts of this study.

Ravaut and Rabeau. VIRULENCE OF THE CEREBROSPINAL FLUID IN CASES OF GENITAL HERPES. [C. R. Soc. Biologie, December 17, 1921.]

The intense nervous reaction occurring in cases of genital herpes has been studied by these observers now for a number of years. Cerebrospinal fluid examination of twenty-six patients showed a reaction of

variable intensity in twenty-one of them. In one case the fluid was actually turbid. They have now endeavored to ascertain whether the fluid in such cases is virulent. They inoculated the cerebrospinal fluid of five different patients suffering from genital herpes on to the scarified cornea of five rabbits. The results of one of these experiments is recorded in detail. For fifteen days after the inoculation the rabbit showed no sign of illness. The local lesion disappeared without leaving any trace. On the fifteenth day, however, the animal commenced to manifest certain nervous changes, such as deviation of the head to the homolateral side, falling over to the same side, and convulsive movements of the limbs. After this phase these phenomena decreased and the rabbit gradually wasted till it died, forty-three days after the inoculation. The brain, which was examined by Levaditi, showed in the region of the mid-brain the perivascular collections of cells so typical of encephalitis in the human subject. A small abscess was found at the level of the aqueduct of Sylvius. Cultures made from the brain were unfortunately contaminated so that a further passage experiment could not be made. The fluid from one of the herpetic vesicles of the same patient when inoculated into another rabbit produced keratitis on the third day, followed by encephalitis on the seventh day. The conclusion seems to be justified that the same virus as is present in the vesicles is also probably present in the cerebrospinal fluid of patients suffering from genital herpes.

Rudolf. BABINSKI'S REFLEX. [Journ. of Neurol. and Psychopath., February 1922.]

Following the suggestive work of Brouwer (*JOURNAL OF NERVOUS AND MENTAL DISEASE*), this paper deals with the phylogenetic significance of the plantar response in man. A large number of animals were tested for purposes of comparison. It was found that the plantar response is of the plantar flexion type in lower animals, whereas in the primates it is of the dorsiflexion or extensor variety. In the human infant there is a period, consisting of the first week of life and to a certain extent the second also, in which a flexor plantar response is often present; but this infantile flexor response changes later into the normal extensor plantar response of the child. This change need not occur in both feet simultaneously, and female infants obtain the extensor plantar response at an earlier period than males. The same primitive flexor response may occur in certain cases of total transection of the adult human cord. Therefore as the animal scale is ascended the type of response varies, for the lower creatures give no response at all, the higher an extensor reflex, whilst adult man gives a flexor plantar response. There is a similar succession of flexor, extensor, flexor in the development of the average individual, for the infantile flexor response changes to the extensor response of the child, and this again to the normal flexor result of the adult. The last is an indication of the highest

degree of evolution, in which greater control over the lower centers of the cord is exercised by the brain. These observations lend support to the view that the extensor plantar response of infants and of spinal lesions in man is an atavistic phenomenon dependent on the prehensile toe of our arboreal ancestors.

Wilson, S. A. Kinnier. DECEREBRATE RIGIDITY IN MAN. [Brain, Vol. XLIII, Part III, 1920. B. M. J. Ed.]

It has been thought and even stated in some quarters of recent years that medicine can expect to learn little more from purely clinical observations. Neurology, however, is fortunate in being a branch of medicine in which original clinical observations continue to lead to great increase in our knowledge of the functions of the central nervous system as a whole. And we are proud to be able to say that in this field British neurology can fairly claim a prominent place. Kinnier Wilson's recent contribution is a shining example, if such were needed, of the value of careful and prolonged clinical study of symptoms which, though familiar to many, have hitherto eluded satisfactory explanation. He has drawn a luminous comparison between the experimental decerebrate rigidity of animals (following transection of the brain stem through the mesencephalon) and certain cases in man "in which there is evidence of withdrawal of cortical control in the form of unconsciousness or semiconsciousness coupled with the appearance of tonic rigidity of the trunk or limbs, with exacerbations in the form of tonic or postural fits." The first group of cases quoted are examples of decerebrate rigidity combined with tonic fits, and pathologically comprised a tumor of the frontal lobe with hemorrhage into the ventricles, a tumor of the mesencephalon, general septic meningitis with some internal hydrocephalus, a case of cerebral hemorrhage with extension into the ventricular systems of the brain, and a case of tuberculous meningitis with moderate internal hydrocephalus. The description of these cases is very full and vivid, and the author points out how the tonic posture was maintained, while the actual fits were an accentuation, so to say, of the already existing decerebrate position. The absence of any clonic element in the fits is emphasized. Tonic or "cerebellar" fits, as they were originally called, may occur without the maintenance of the decerebrate posture, and a very striking example of such a condition is given in full. Their association with hysteria is also pointed out, the position of opisthotonos with extension of the legs and extension and pronation of the arms being a well recognized phenomenon. Of particular clinical interest are those cases which exhibit a unilateral or fragmentary decerebrate attitude in one limb only. The so-called decerebrate attitude of the head may be thus explained. The extreme pronation of the hand which is found, for example, in athetosis or chorea is held by the author to be but a fragment of the complete picture of the decerebrate posture. In explaining

the pathogenesis of this condition tribute is paid to the theory of "cerebellar influx" propounded as long ago as 1877 by Hughlings Jackson. The numerous experimental researches of Sherrington, Graham Brown, Weed and others, however, point to the conclusion that the mesencephalon is the principal site responsible for the maintenance of the decerebrate posture; and the red nucleus is credited with particularly important functions in this respect. We are promised a further study on the part played by the basal ganglia in the regulations of these functions. No student of neurology can afford to miss this communication, to which a full bibliography is attached; and further contributions will be awaited with interest.

Gelpke, H. CONGENITAL DEFECTS OF BASE OF SPINAL CORD. [Monat. für Kinderh., November 1920.]

Myelodysplasia and spina bifida may be associated, but in that case they are coördinate phenomena of the same disturbance in development; neither is subordinate. Fuchs' "rudimentary forms of spina bifida" is regarded here as a sign of degeneration that gives a valuable indication of the existence of an occult spina bifida.

Roccavilla, André. REGIONAL HETEROMORPHOSIS OF THE SPINE. [Rev. Neurologique, January 1921.]

Abnormalities of the vertebræ which give rise to nervous symptoms and which often remain wrongly diagnosed until X-ray examination is made are here discussed. Certain levels, namely, between the cranium and the atlas and between the cervical and dorsal, dorsal and lumbar, lumbar and sacral, and sacral and coccygeal vertebræ are more prone to be involved than others. The characters of the vertebræ at these levels are unstable. Thus the seventh cervical often assumes some of the characters of the first dorsal. The clinical importance of these malformations varies. Occurring at the dorso-lumbar junction, they may give rise to no symptoms. At the cervico-dorsal junction, the signs of cervical rib are well known. That changes at the atlanto-occipital and the sacro-coccygeal joints are sometimes responsible for neuralgic troubles is not so well recognized. The signs of lumbo-sacral malformations are even less known and it is with these that the writer specially deals. Here the fifth lumbar vertebra assumes sacral characters, it undergoes sacralization, the transverse processes spread out in butterfly form and may fuse with the iliac crests, giving a striking radiographic picture. In consequence, certain nerves suffer, in particular the fifth lumbar and less frequently one or two nerve roots above and below. Pain is the chief symptom. In slight cases it is lumbago-like, in others, it is paroxysmal, intense and radiating. Tender points may be found where the fifth lumbar and first sacral roots emerge. Strips of cutaneous anesthesia or hypoesthesia running down the back of the leg from buttock to peroneal region, or in the distribution of the fifth lumbar

root may also be discovered. Since this abnormality is found to occur in 4 per cent of all persons, its diagnosis is important. Rheumatism, Pott's disease, sciatica and stone in the ureter come to mind as possibilities until X-ray examination determines the diagnosis. Treatment is unsatisfactory. In slight cases instructions are given to avoid movements which aggravate the malformation. In worse cases immobilization and surgical measures (removal of the offending bone) must be considered.

De Stefano. FAMILIAL SPASTIC PARAPLEGIA. [*Pediatrics*, October 1, 1920, XXVIII, No. 19.]

In this clinical paper the author reports upon five cases in two families which he has seen within the year. Two other members of one of the families are known to have suffered from the same. The paraplegia developed at the age of sixteen months, two and one-half, four, five and six years respectively in his five cases. A history of syphilis was present in both families. Physical and mental deterioration was progressive, one child in each family becoming demented or was born idiotic. Intraspinal specific treatment, he thinks, might be given a trial in such familial cases if seen before the meningeal reaction has brought about degeneration.

Hamburger, W. PREVENTIVE INOCULATION AGAINST RABIES. [*Nederland. Tijdschr. v. Geneesk.*, February 18, 1920.]

In 1919, nineteen persons received preventive inoculation against rabies at the Utrecht Serological Institute. All came from eastern provinces, and as Holland had been free of the disease within recent years it had probably been introduced from Germany. The results of the inoculation had so far been good. Of the 19 cases which had been inoculated from a few hours to twenty-nine hours after being bitten none had contracted the disease. Paraplegia and rectal and visceral paralysis, which have been described after antirabic inoculation, were not observed.

Stewart, F. W. THE GANGLION CELLS OF THE NERVE TERMINALIS. [*Jl. Comp. Neur.*, August 15, 1920.]

The origin cells of the *nervus terminalis* of the white rat arise in a proliferation from the septal aspect of the olfactory sac, including the epithelium of the vomero-nasal organ. The tendency to regard these cells as vegetative is not supported. Sections of embryos showed definite proliferation from the epithelium of the olfactory sac, quite independent of any trigeminus components. The facial nerve was seen to be accompanied by cells which give rise to the sphenopalatine ganglion and it might justly be assumed that addition to the ganglion cells of the nasal cavity would be essentially a continuation of this same migration. No trace was found by Stewart of any cells from the sphenopalatine

ganglion into the nasal territory and neither has the study of trigeminal branches been productive of any result. Cells from the cervical sympathetic have been followed along the internal carotid nerve and the great deep petrosal nerve as far as the Vidian nerve, but no further. Similarly, no evidence could be obtained as to their origin from the neural tube; in fact, any growth appears to take quite the opposite direction, no contact of ganglion cells with brain wall occurring until late stages, when cells are carried backward with growing nerve rootlets. The writer sees no reason for reversing his earlier conclusions that the derivation of the ganglion cells of the *nervus terminalis* is from the epithelium of the olfactory sac.

Kretschmer. FAMILIAL CEREBRAL INFANTILE PARALYSIS. [Deut. med. Woch., November 4, 1920, XLVI, No. 45.]

This clinical record of familial cerebral infantile paralysis details the histories of three brothers, aged twenty, sixteen and fifteen, respectively. A sister, aged fifteen, is well. The process became manifest first at the age of twelve in each boy and spastic paraplegia (tetraplegia), talipes and pes excavatus, partial atrophy of the optic nerve, retardation of speech and more or less marked signs of dementia developed slowly. All three brothers were normal physically up to twelve years. The oldest was normal mentally; the two younger were not so good. The hypothesis of Gowers is used in partial explanation.

Myers, Alonzo. EARLY, INTERMEDIATE, AND LATE TREATMENT OF ONE THOUSAND CASES OF INFANTILE PARALYSIS.

The problem of the treatment of infantile paralysis is best learned by observations of neglect and the results of treatment as observed during a large dispensary service in the city hospitals, particularly by those making a specialty of orthopedic conditions. In my service at the New York Orthopedic Dispensary and Hospital, and during visits to Johns Hopkins University at Baltimore, the University of Pennsylvania at Philadelphia, Harvard University at Boston, and during my thirty-three months of war surgery on the orthopedic services of the French hospitals while associated with Dr. Jos. A. Blake in Paris, I have observed over one thousand cases of infantile paralysis in its various stages—some that had received no treatment, others that had had interrupted treatment, and those that had had careful treatment, over a long period. I shall not give a tabulated list of the different regions paralyzed, but rather a general résumé of patients as they come to hospitals for treatment. These cases as they go from one dispensary to another become so anxious, they are prone to accept suggestions and advice from either the laity or the profession, and are too willing to try anything with the hope of cure. Unfortunately, many physicians offer bright hope of cure with electricity and the orthopedic surgeon sees the horrible results in deformity and disability that may have been

prevented by absolute rest. In the early or acute stages as seen during the 1916 epidemic in New York City, the best results were obtained with plaster-of-Paris or braces, and rest in bed.

After the painful stage is past, general massage and muscle training is started, and this must be done under the absolute direction and supervision of the orthopedic surgeon. Overstretching muscles that are weakened but are not paralyzed must be carefully guarded against. These are the muscles that are reported as cured paralyzed muscles, but in reality were only weakened muscles. Every muscle is essential in reconstruction, therefore every precaution must be taken toward this end. When the period of immobilization has passed and all deformities have been corrected, braces are indicated to hold in the corrected position. This period varies, and from six months to a year should elapse before strain, such as weight-bearing or overzealous exercises, are allowed, and the patient should be carefully watched for an indefinite period. In the intermediate stage, some cases have been neglected, others poorly treated, and here is where we see the deformities of infantile paralysis either from neglect or ignorance. Correction and braces are the procedures, operating when the deformity cannot be overcome with stretching under general anesthesia. Slight contractions may be corrected by stretching, while more extensive contractions require tendon lengthening and tendon transplanting. In the flail joints and the deformities due to the stretched ligaments and disturbed bony relations, we resort to immobilization by various methods, as the arthrodesis of Jones and Davis in the foot, Myers in the knee (using the patella wedge) osteoplasty of the spine after Hibbs. Flail hips are most unusual, I having seen one only in a thousand cases; an arthrodesis, according to Albee, will suffice here. When all deformities have been corrected, it is of prime importance to hold them with braces and of secondary, if not of equal, importance to begin muscle training. All cases should receive massage daily and this should be followed by muscle training. Complete knowledge of anatomy and physiology is necessary to obtain an accurate diagnosis before outlining the muscle training exercises.

In the late stage, when atrophy is present and marked deformity due to the constant stretching of the unparalyzed muscle, massage and muscle training are particularly important, since improvement has been noted in cases after thirty-six years' duration, as reported by Lovett of Boston, and as I have observed in my dispensary treatment, where patients have wandered from one dispensary and clinic to another over long periods, after which systematic massage and muscle training resulted in marked improvement, both functionally and esthetically.

The desired results and treatment of infantile paralysis as detailed in this paper may be summarized as follows: First, in the acute stage, immobilization either with plaster or braces, rest in bed, and active treatment after tenderness has disappeared; second, massage and muscle

training; third, stretching, an operative correction of deformity, followed by the application of braces; fourth, constant supervision and attention over a long period of time. [Author's abstract.]

Mittelstädt, W. PARALYSIS OF THE ABDOMINAL MUSCLES IN POLIO-MYELITIS. [Zschr. f. d. ges. Neurol., Vol. LVIII, 1.]

Report of four cases. The first showed the scoliosis already observed elsewhere on standing as result of the paralysis of the recti abdominis muscles. For this the explanation given by Strasburger is accepted, preponderance of the depressor of the anterior pelvic wall as a result of the prolapse of the recti abdominis which with the gluteus maximus act as levators of the anterior pelvic wall. The same case in examination of the active boat-like contraction of the abdomen, which represents a special function of the transversus, showed a ring of contraction somewhat above the navel with arching of the portions of the abdomen above and below that ring, to be explained from the partial compensation of the corresponding portion of the transversus ninth dorsal. The symptomatology of the poliomyelitic paralysis of the abdominal muscles as it is variously extended is discussed in detail. The segmental innervation of the abdominal musculature first advanced by Ibrahim and Hermann has since found confirmation. Contrary to Oppenheim's opinion segments are to be considered also above eighth dorsal.

Wilson, George. SPINAL AND SPINOBULBAR TETRAPLEGIA OF ACUTE AND SUBACUTE ONSET. [Jl. Am. M. A., March 11, 1922.]

As examples of paralysis of all four extremities due to thrombosis of the anterior spinal artery, three cases are reported. In the first case there was occlusion of the anterior spinal artery at about the junction of the bulb and the spinal cord, thus involving the pyramids and the lemniscus, which lies immediately behind. The second case is an example of the same condition as that manifested in the first case although in this patient the area of involvement went lower into the spinal cord because there was atrophy of the muscles of the left shoulder girdle. The case illustrates the rare occurrence of an involvement of the pyramids as they are decussating. In this man, although all four extremities were equally paralyzed at the onset and for months afterward, at present the right upper and the left lower extremities show by far the greatest improvement. The third case is one of cervical myelitis probably due to occlusion of the anterior spinal artery or anterior median spinal artery. In this case the symptoms presented on examination indicated a lesion lower than in the other two cases. This case appeared to be one of occlusion of the anterior median spinal artery affecting chiefly the lower cervical cord. The anterior horn cells were involved, as shown by the atrophy of the forearms and the hands. The anterior horn cells in the lower part of the cord were also involved, but this may have been a process that occurred after the original

thrombosis. The absence of pyramidal tract symptoms and the preservation of sensation are due to the fact that the anterior median spinal artery does not supply the posterior columns or the pyramidal tracts. If hematomyelia occurs in the upper cervical spinal cord, there may be paralysis of all four extremities as shown by two cases cited. Cervical myelitis originating in the course of acute infections or occurring secondary to the disease of the blood vessels produced by syphilis may cause tetraplegia as in one case reported. Wilson has also seen one case in which paralysis of one arm and both legs came on suddenly, followed in a short time by paralysis of the other arm, the condition being due to Pott's disease. The cases presented are not only of interest from the etiologic point of view and from the location of the lesions, but also of importance because of the recovery which most of the patients showed. Those cases with the most favorable prognosis are those of syphilitic origin in which the treatment is pushed. The two patients with hematomyelia improved remarkably, one of them making a complete recovery and the second patient regaining more power as time goes on. The woman who developed tetraplegia during the course of epidemic encephalitis and who was paralyzed for months made a complete recovery.

Bordier, H. NEW CONCEPTIONS ON TREATMENT OF INFANTILE PARALYSIS.
[Arch. Radiol. and Electroth., 1921, XXVI, 215. Med. Sc.]

The author, in agreement with others quoted, does not believe that the disease need necessarily lead to complete crippledom if properly treated. He reviews the appearance of the cord in the portions affected. In his view the logical therapeutic indication is to act on the medullary lesion by radiotherapy, as he asserts that it must have the effect of reestablishing the cells incompletely destroyed, and of arresting the progress of the poliomyelitis. Treatment must be undertaken as early as possible. The author employs a series of three consecutive radiations once a month with 5 mm. Al screen. The second point is to combat the lowering of the temperature of the affected limb, and for this purpose diathermy is the most efficacious means: various other methods tried are enumerated. The technique is described. Treatment is continued for four or five days, after which the limb retains the warmth; this is the time to begin electrical treatment. If necessary, diathermy is repeated. The electrical treatment may have to be continued for months or years.

Dubs. SIMULATION OF APPENDICITIS BY ACUTE ANTERIOR POLIO-MYELITIS. [Schweiz. med. Woch., March 17, 1921.]

This interesting clinical study records the histories of three cases in which the symptoms of acute anterior poliomyelitis simulated those of acute appendicitis so closely that laparotomy was performed. A few days after the operation flaccid paralysis developed. While admitting that these cases showed blunders on the part of the surgeon, the author

insists that the rule of operating on acute appendicitis at the earliest possible stage is excellent, although it entails the inclusion of a certain number of mistakes in the surgeon's list of laparotomies. These mistakes must be regarded as far less serious than deferring operations for appendicitis till the diagnosis is invariably placed beyond doubt. In the author's cases the local signs conformed in a remarkable degree to the well recognized clinical picture of appendicitis; there was marked tenderness with rigidity at McBurney's point, and sudden release of pressure on the abdomen provoked lively pain. But, he admits, there were certain features of the general symptomatology in the first case which should have made him give pause. The patient, an unmarried woman of twenty-one, complained of general lassitude, headache, and excessive perspiration. Her temperature was high from the outset (102.2°). The author subsequently remembered that he had never seen a patient suffering from acute appendicitis complain of headache and excessive perspiration, and he considers a high temperature early in the disease as a rare concomitant of acute appendicitis. He also refers to the simulation of acute appendicitis by encephalitis, a case in point being recently recorded by Seidel (*Muench. med. Woch.*, No. 7, 1921, p. 219).

Semerak, C. B. CENTRAL NERVOUS SYSTEM IN BOTULISM. [*J. Infect. Dis.*, 1921, XXIX, 190. *Med. Sc.*]

An account of the changes in the central nervous system of a girl aged seventeen who died with symptoms suggestive of, but not definitely proved to be, botulismus. In favor of the diagnosis were the facts that several persons were affected after consuming the same smoked ham and salted pork, the similarity of the clinical course and symptoms and their correspondence with changes in the central nervous system, the exclusion of trichinosis, or ptomaine poisoning, and also the fact that the lesions did not correspond to epidemic encephalitis or to other known forms of the disease. The lesions were confined to the vascular system of the central nervous system, thrombosis in arteries and veins being the initial change, followed by ischemic necrosis, and later by inflammation. According to the author the poison had no direct action on the nerve-cells, the regressive changes being secondary and due to disturbances in the blood-supply. The ganglion cells of origin of the motor cranial nerves are always involved because their blood-supply comes from terminals of branches of the vertebral arteries, which appear to be the seat of predilection of the thrombosis.

Sabatucci, F. PARAPLEGIA IN MALARIA. [*Il Policlinico, Sez. Prat.*, February 16, 1920.]

Malarial myelitis is not unknown but is rare and the two case reports here recorded are of interest. Two soldiers, contracting malaria in Albania, began to suffer from paresthesia and weakness in the lower limbs. Intermittant claudication and finally complete paraplegia with

sphincter disturbance, bedsores, and trophic ulcers then developed. Loss of all kinds of sensibility, most marked in the distal segments. Paraplegia, at first flaccid, later spastic, was present. Under vigorous quinine treatment improvement took place gradually until only slight spastic paraparesis remained in one case; in the other the symptoms were a little more pronounced.

Pirone, R. CULTIVATION OF VIRUS OF HYDROPHOBIA. [*Riforma med.*, 1920, XXXVI, 782.]

Investigations of an incomplete character which were made before the war with the purpose of controlling the results arrived at by Noguchi. The author was unable subsequently to repeat and complete his experiments, but he thought it useful to publish them without further delay as they confirm the possibility of cultivating the virus of hydrophobia by Noguchi's method. In the cultures of two different series of experiments bodies similar to those described by J. Koch were found. [Da Fano.]

Marchand. HISTOLOGICAL LESIONS IN RABIES. [*Bull. et Mém. Soc. Méd. des Hôp. de Paris*, December 30, 1920.]

The histological picture found in a man dying six weeks after dog bite is given as follows: No naked-eye lesions were seen. Histologically the cerebrum was intact, the lesions being confined to the medulla oblongata, where they consisted of perivascular inflammation chiefly affecting the vessels of the floor of the fourth ventricle, very slight inflammatory areas in the other bulbar regions, and definite chromatolysis of the cells of the cranial nuclei. There was an absence of Negri bodies.

Spatz, H. DEGENERATIVE AND REPARATIVE PROCESSES AFTER EXPERIMENTAL INJURY OF THE SPINAL CORD. [*Zschr. f. d. ges. Neur.*, Vol. LVIII, 327.]

Spatz follows Cajal in insisting that one must consider the degenerative process which begins at the wound and extends into region of the cells of origin as one and the same process. There is no line to be drawn between the "traumatic degeneration" in the central stump and the acute "retrograde degeneration." He compares the primary change to a wave which forces itself out from the location of the lesion, the extent of its attack being due to the strength of the stimulus and the remoteness of the cell from the starting point of the wave. As the primary changes begin in the axis cylinder, the "primary irritation" of Nissl takes place in the cell of origin, the change in both cases consisting in the swelling of the indifferent protoplasm, the hyoplasm, by which the products of differentiation which are present are forced toward the periphery. In the case of the ganglion cells this affects the nucleus and the tigroid substance. These primary changes play themselves out in the central stump and in the portion of the peripheral

stump closest to the wound. The secondary degeneration of the entire peripheral portion of the neuron must be sharply distinguished from these primary changes. They are a direct result of the interruption of the continuity and their extent is therefore quite independent of the degree of the lesion. The reparation process will be found distinguished by different zones with a characteristic histological picture for each one. Directly adjacent to the wound the disintegration of all the elements of the nervous tissue constitutes a "zone of débris." Here the neuroglia is insufficient for activity so that the mesodermal tissue alone, not chiefly the autochthonous tissue but that which has grown in from without, must perform the functions of removal and repair. Further removed are the "zones with lacunæ" in which original elements are found intact between the altered neuron elements and the neuroglia is sufficiently preserved to take up its activity in participation with the mesodermal elements. Spatz calls attention to two more or less analogous types of injury represented by the zone of débris and zone with lacunæ which have been demonstrated in injuries of the spinal cord in man (Marburg), in concussion of the spinal cord (Schmaus), in myelitis in man and experimental myelitis in animals (Mager, Lotmar). [Author's abstract.]

Jorge. DISLOCATION OF VERTEBRA WITHOUT SYMPTOMS. [Sem. Méd., October 21, 1920, XXVII, No. 43. J. A. M. A.]

Jorge's roentgenograms show complete dislocation forward of the fifth cervical vertebra while the sixth is still in normal position, but there have been no symptoms on the part of the spinal cord during the six months since the fall causing the dislocation. Movements of the neck are painful, and Jorge is planning to have the man wear a collar to support the head as in a hammock and induce the necessary hyperextension. If this can be realized, the man of thirty-five may be able to regain partial earning capacity at least. If symptoms on the part of the spinal cord develop, surgical measures will of course be indicated at once.

Marshall, H. W. SPINAL INJURIES. [Boston Med. and Surg. Journ., 1920, 182.]

Vertebral lesions in which there is no cord injury are alone dealt with in this clinical paper reviewing thirty cases. Radiography allows positive diagnosis of vertebral fracture to be made in cases which would formerly have been labelled "back sprain." Such injuries are not permanently crippling, and the patients frequently develop a high degree of physical efficiency, sometimes within a few months. Mechanical appliances are required very commonly for considerable periods of time, and indications are given as to the best kind of apparatus to apply. A very light flexible steel pelvic band with light steel uprights is his pet brace. The inlay bone graft is not favorably thought of. It can have

but little effect in relieving a crushed vertebral body from compression, he thinks. Operations are not uniformly beneficial, and fair or excellent recoveries are so common without surgical intervention in healthy young adults that bone grafts are rarely warranted.

Floeckinger. DECOMPRESSION OF SPINAL CORD IN TUBERCULOSIS. [Amer. Journ. of Surg., March 1920.]

In this clinical paper, simple decompression is the operation of choice to prevent complete destruction of the cord by pressure. When the symptoms are still those of compression, without degeneration, the prognosis is good. Nothing more than simple decompression is necessary. The open scooping operation is contra-indicated. These cases should not be drained. Should the granuloma be incised during the operation the débris should be carefully scooped out, the wound dried, mopped out with lysol or phenol solution, and then dried again and sprinkled with powdered iodoform. Improvement is gradual after operation and plastic jacketing, and the results of tuberculin treatment are most promising provided it is never given more often than once a week, the daily fluctuation of temperature being used as a guide to the opsonic index. Removal of the spinous processes and the arches of the vertebræ does not affect the strength of the spinal column materially.

Prat. TRAUMATISM OF THE SPINAL CORD. [Rev. Méd. del Uruguay, December 1920, XXIII, No. 12.]

Two cases of uncertain diagnosis are here reported upon in which there was extravasation of blood in the cervical portion of the spinal cord following trauma. The C. S. F. was blood stained in both; in the first case the head could be moved; radioscopy was negative in both. The only sign in one case was the semierrection of the penis. There was marked bradycardia 22 beats to the minute. Faradic and galvanic electric tests seemed to indicate that the spinal cord had not been totally severed, and necropsy confirmed that in both the lesion was a 2 cm. in extent hematomyelia of the gray matter.

Lenep, H. von. SPINAL CORD TUMORS. [Deut. Zeit. f. Chir., November 1920.]

This clinical and historical study compares three cases of his own with 153 which he has collected from the literature. Thirty-three per cent were reported as cured and 15 per cent improved, but 33 per cent died and in 11 per cent no benefit followed the operation. Of 25 with intramedullary tumors, 28 per cent were materially improved and 52 per cent died. Sixty-four intradural tumors were reported as 65.6 per cent cured or materially improved, with 25 per cent mortality. In 30 cases of vertebral tumors, 23 per cent were improved and 50 per cent died, including all the carcinoma cases and 3 of the 12 sarcoma cases.

BOOK REVIEWS

Levinson, Abraham. CEREBROSPINAL FLUID IN HEALTH AND DISEASE. Second Edition. [C. V. Mosby Company, St. Louis. \$5.00.]

We found the first edition of this work admirable. The second is even better. It has been enlarged and revised and constitutes one of the very best manuals for all those interested in the study of the cerebrospinal fluid.

The chapter on cerebrospinal fluid in various diseases could be amplified somewhat to advantage. The presence of blood in certain cases of epidemic encephalitis is not mentioned, nor are Ayer's interesting findings of the colloidal gold reactions in multiple sclerosis. Kaplan's valuable monograph is not referred to, and Sicard's important researches in cytology could have been expanded with profit.

Bonnier, Pierre. DÉFENSE ORGANIQUE ET CENTRES NERVEUX. Nouvelle Edition. [Félix Alcan, Paris.]

This is a posthumous revised edition of Bonnier's ideas, done by his widow. They have been before the scientific world for a number of years. We do not know whether the extraordinary results claimed by him have been extended or verified.

He maintained that in the bulbar and related central synapses of the trigeminal nerve terminals located upon the skin of the face and within the mucous membranes of the nose there had been elaborated a highly organized group of defense mechanisms against a vast number of bodily disequilibrating forces. By proper stimulation of these terminals, cauterization chiefly, which must be done with great discrimination, this central defense apparatus could be so played upon as to bring about very striking therapeutic results in a vast variety of syndromes. Sometimes an asthma departed after a single cauterization, three or four applications cured many things, while in one instance it required fifty applications to finally reach the digestive centers and to cure a chronic constipation with a continuous migraine. The cauterization of the average rhinologist is a brutal thing, for these purposes; the touch must be just light enough to set in operation those reflex forces of defense which will restore the equilibrium of the disorganized centers. Miracles he calls them. Many of these miracles were wrought in the Rothchild clinic, from which many similar miracles have issued of late from very light doses of the endocrines.

We are reminded of the numerous miracles produced by the eye

muscle cutters of the 80's in the United States and the clitoris cutters of the sixteenth century and the everlasting "clyster" cures.

In his "Action directe sur les centres nerveux" (Alcan, 1913) Bonnier gave us a series of rough charts locating these centers and the spots on the nasal mucous membrane that should be cauterized.

Among the disorders cured by this method are reported melancholia, homosexuality, obsessions, amenorrhea, enteritis, dysmenorrhea, impotency, frigidity, priapism, neuralgias, cancer pains, impetigo, enuresis, in healthy and in imbecile children, constipation, pronasitis, otorrhea, hemorrhoids, varices, acne, emphysema, cirrhosis of liver—but enough. It does too much.

Foote, Theodore Clinton. THE SOURCE OF POWER. [Williams and Wilkins Company, Baltimore.]

This is a valiant book of 170 pages. It consists of a series of asseverations about mind, body, will, intellect, sensation, consciousness, the subconscious, telepathy, faith and other well known words, but really tells nothing. None of the statements on the jacket about the book fit in at all with the reviewer's opinion.

Roback, A. A. BEHAVIORISM AND PSYCHOLOGY. [University Book Store, Cambridge.]

Psychiatry has always been a type of behavioristic psychology. At least it started so with Democritus; for behind his explanation of hysterical phenomena one can perceive he was a dynamist and a behaviorist. When Protagoras was eclipsed by the sophistries of Plato psychiatrists became absolutists and intellectuals for many a century and only began to come back to the behaviorism of the Sophists through Paracelsus, Plater, Stahl and Reil.

Fortunately a new behaviorism has arisen in contemporary psychological circles and psychiatry again hopes to be understood and to be assisted somewhat by the faculties.

The great war drew into its delirious confusion many school psychologists. Many of them learned much about the behavior of human beings which their terminological hair-splitting had skillfully concealed from their vision. The new behaviorism took much from these lessons. How much has remained, no one can prophesy.

The present volume would examine into this newer behaviorism of the schools; how far it applies to psychiatry and what the author may contribute we shall see.

In the first place he employs his narrow logical(?) intellectualism to show that because behaviorists have used so many concepts they cannot all be true hence none of them possess much sense. Physiologists have used the *term* behavior, therefore it can have no psychological significance; it becomes *verschommen* he says, as if physics and chemistry had no relation to life, and to living processes. He then gives a venomous thrust at Holt, as a protagonist of the Freudian wish hypothesis; expanding it to subvert Watson in his contribution to "Behavior and the Concept of Mental Disease." While the reviewer may agree that Watson was somewhat on the bias as to

what mental disease really connoted, still his naïve ignorance of the details of psychiatry did not too seriously impair the validity of essential behavioristic interpretations. They were at least better efforts to get in touch with reality, even if "impatient," than the dogmatic terminologies of the academic psychologists who are really crassly ignorant and smugly intolerant of the psychoanalytic findings. If it came to a *show-down* between the author's antithesis between the "hoss doctor" and the "man doctor" (p. 151) the reviewer would incline to an intelligent veterinarian rather than to an autistically trained "man doctor," solely on the behavioristic interpretation. If "man doctors" could take a preliminary "hoss doctor" training they would be better psychiatrists, as Kempf has abundantly demonstrated. Boas has frequently emphasized that the "primitive" is not such an ass as the university instructor would lead us to suppose. Roback implicitly acknowledges this in his Chapter XII on Behaviorism and Religion.

In Chapter XIII the author comes into his own. He is essentially a believer in *tests*. Intelligence, as determined by Binet, or Terman, or Roback, is really what constitutes the real thing, struggle as he may with its insuperable difficulties. Paton has related, with amusement, how a dementia precox individual passed 100 per cent with his Princetonian intellectual tests. Are we to conclude from such a criterion that when we all become schizophrenics we shall be eligible to the honorary degree of D.Sc.?

And now to our "moutons." Part IV, Outlook of the Conflict, as the author terms it; What is the future of "Behaviorism"? These are "weighty" questions, which must not be answered with an "*unbiased*" mind. Where such a mind will be found the author does not tell us, much less conceive of, save in terms that *my* doxy is "orthodoxy" and the other fellow's "heterodoxy." "Behaviorism" is not my—Roback's—doxy, hence it is "heterodoxy." "Thee is also queer" is the final analysis of this more than intellectualistic effort to enhance the author's own point of view. So long as disease still remains unconquered, so long as man still carries a bunch of keys in his pocket to prevent himself from being robbed; so long as jails and judges still are present in the community the type of thinking that the author's presentation would put in the "seats of the mighty" will remain ineffectual to cure the ills which they foster and represent. "Thus saith the preacher," moron though he be as judged by the Platonic standard.

In the fashion of our newspaper dramatic critics, a purely negativistic performance with no constructive outlook.

Bianchi, Leonardo. THE MECHANISM OF THE BRAIN AND THE FUNCTION OF THE FRONTAL LOBE. Translated by J. H. MacDonald. [E. S. Livingstone, Edinburgh. 21s.]

Of modern neuropsychiaters Bianchi stands preëminent. Not only is he conversant with the researches of modern neuroanatomy, of psychology, of social activities, but as minister of public instruction in Italy, he has combined more than perhaps any contemporary

scientist the full gamut of possibilities. Laboratory worker in structure, experimental physiologist, observer of national, local and individual behavioristic reactions, as individual educator, director of an asylum, and as governmental official directing concerted mass action over educational efforts, he has combined more activities than any figure with which the reviewer is acquainted.

With this foundation, and as a product of his later years, he gives forth this volume. Limited though it may be chiefly to experimental work upon lower animals, there runs through it all the richness of his manifold activities and stamps it as a production of transcendent importance.

Fortunately he has found an able translator. MacDonald's work we can first commend. This is not the only service he has rendered to the cause of international sympathy and affiliation. He has made it possible to get the ideas of a master in his field and for which the world of serious minds must be thankful. In this endeavor the publisher has done his part most admirably.

Concerning the motif of the book itself, Bianchi sets forth the hypothesis, with which few can at present disagree, that the frontal lobes represent the organic substratum of the highest integrative possibilities of human social relationships. It is an organ—the most advanced organ—whose functions are best described as “intellectual.” He does not deny the interrelationships of other cortical, subcortical, striatal, medullary, spinal or peripheral mechanisms.

The organism as a unit with definite teleological trends, he accepts. He advocates the idea that these are made most effective through frontal lobe activities. Not one frontal lobe but both are concerned in this integration. For Bianchi there are no “silent areas.” Such “silence” is only ignorance. In support of this general thesis, which has really been accepted by most workers, he adduces a series of experimental observations on trained lower animals. This mode of approach has its values. There are others. He is not unacquainted with these.

Altogether this work is to be most enthusiastically commended in its field. It supports the contentions of the evolutionist. It amplifies the range of experimental methods as a means of solving many important problems of human conduct. It gives an objective basis for fundamental conceptions of human behavior. It is a masterpiece of research. We find only one defect in it. Shepard Ivory Franz, one of America's most illustrious workers in the same field, is consistently quoted under the name *Sheperd*, rather than *Franz*.

Barker, Lewellys F. CLINICAL MEDICINE. [W. B. Saunders Company, Philadelphia and London.]

Dr. Barker's Tuesday clinics at the Johns Hopkins Hospital are here printed in a volume of 600 pages. They are an interesting collection. For the neuropsychiatrist the book will prove of special interest as at least half of it is devoted to most illuminating case reports and discussions of nervous and mental syndromes.

NOTES AND NEWS

Dr. D. J. McCarthy, Chairman of the Division Council of the Pennsylvania Mental Hygiene Society, is organizing a state-wide representation which will include laymen with particular interest in mental hygiene as well as additional representatives from the medical profession. The Medical Director of the Division, on duty since January 1, 1924, at 419 S. 15th St., Philadelphia, is Dr. Alfred J. Ostheimer, M.D., L.R.C.P. (London) M.R.C.S. (England). He was formerly associated with Dr. D. J. McCarthy in the practice of neuropsychiatry, served as a Lieutenant-Colonel in the Medical Corps of the army during the war, and since, has been in charge of the ex-service men suffering from mental and nervous diseases in this, the Third District of the United States Veterans Bureau.

H. N. MOYER

RESOLUTION OF CHICAGO NEUROLOGICAL SOCIETY

WHEREAS, it has pleased God in His infinite wisdom to remove from us our fellow member, Harold N. Moyer; and

WHEREAS, the Society in him has lost one of its founders and guiding spirits, a faithful attendant and contributor to its proceedings, who served twice as its president, who gave liberally to his fellow members both of his professional knowledge and his personal charm; and

WHEREAS, the insane of the state in him have lost a friend who began his service to them in his youth as physician in a state institution and carried on his activities in their behalf as an educator, reformer, counsellor in legislation, consultant and student; until the last in active attendance as consulting physician to the Cook County Psychopathic Hospital; and

WHEREAS, midwestern neuropsychiatry has lost from its midst a man not only learned and skilled in his specialty but by virtue of his broad interests, culture and literary ability, an unequaled interpreter of neuropsychiatric and medicolegal problems for the public at large whereby he furthered the best interests of mental hygiene; and

WHEREAS, the Society feels that in his death it has lost a friend, distinguished in his chosen field of medicine, beloved for his genial personality, and admired for his personal integrity; be it,

Resolved, that the members of the Chicago Neurological Society express their sorrow and extend their most sincere sympathy to the family and friends of Doctor Moyer; and, be it further

Resolved, that copies of these resolutions be sent to his family, to the Archives of Neurology and Psychiatry, to the *Journal of the American Medical Association*, to THE JOURNAL OF NERVOUS AND MENTAL DISEASE, and to the *American Journal of Psychiatry*, and that a copy be preserved in the Archives of this Society.

Dr. B. F. Alford, of St. Louis, was elected Vice-President of the Central Neuropsychiatric Association instead of Dr. Adler as announced in a recent note concerning this Association.

N. B.—All business communications should be made to Journal of Nervous and Mental Disease, 64 West 56th St., New York.

All editorial communications should be made to Dr. Smith Ely Jelliffe, Managing Editor, 64 West 56th St., New York.

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ORIGINAL ARTICLES

THE INTERSTITIAL TISSUES OF THE CENTRAL NERVOUS SYSTEM: A REVIEW *

BY PERCIVAL BAILEY AND GRACE HILLER

The attention of neurologists has been for so long a time attracted by the neurones, because of their fundamental physiological importance, that the study of the interstitial tissues of the central nervous system has been relatively neglected. There has been a great lack of proper staining methods. The success of the silver impregnation procedures of Cajal and Bielschowsky has led to persistent attempts on the part of many investigators to develop similar methods which would display the interstitial structures with equal distinctness. We may mention the efforts of Perusini, Montesano, Ranke, Snessarew, Tello, Achúcarro, etc.

Of course the method of Golgi (18) was capable of revealing the external appearance of the glia cells, even to their finest ramifications, but it showed nothing of the internal structure and was so uncertain in its action as to be useless for pathologic investigation. Its results on normal tissues, in the hands of Golgi, Cajal, Retzius, Andriezen and others, are still indispensable for the proper interpretation of the results of other methods.

A second epoch in the study of the interstitial tissues was initiated by Weigert (34) with the publication of his selective method for the neuroglia fibrillæ. By means of it, and its numerous modifications and successors (Benda, Mallory, Anglade, Da Fano, Bailey), numerous contributions have been made to our knowledge of the glia in pathological conditions. But by all these procedures the greater

*Surgical Clinic of Dr. Harvey Cushing and Laboratory of Surgical Research, Harvard Medical School.

part of the glia cells are left unstained, and an understanding of what they are doing would be very interesting.

For this reason attempts were made to devise methods for staining the cytoplasm of the cells which would not, like the procedure of Golgi, obscure the internal structure. Held, Eisath, Fieandt, and Alzheimer were among the most successful investigators, and especially the methods of Alzheimer (4) have been fruitful in neuropathologic research. But even Alzheimer himself recognized the inadequacy of the results obtained.

It was not until the publications of Achúcarro (3) and S. Ramon y Cajal (11) that really satisfactory methods were at the disposal of histopathologists. The epochs of Golgi, Weigert and Alzheimer are over. A new epoch has been inaugurated, and up to the present time chiefly exploited, by S. Ramon y Cajal and his brilliant pupils, P. del Río-Hortega and N. Achúcarro, the latter unfortunately dead at the height of his career.

The work of these Spanish histologists is not widely known, since it is published in journals having a very restricted circulation in this country. We have been using their methods for the last two years in the study of so-called gliomas, and at the insistence of our friends have decided to set them down in English, in the form in which we have found them to be most useful, for the benefit of others who may wish to employ them, and an opportunity will be taken to make a few remarks concerning the facts they have revealed in the structure of the interstitial tissues of the central nervous system.

We may distinguish, aside from the connective tissue, four distinct types of cells, which will be discussed in turn, together with methods of impregnation. They are:

1. Neuroglia.
 - a. Protoplasmic.
 - b. Fibrous.
2. Mesoglia(?)
 - a. Microglia.
 - b. Oligodendroglia.

NEUROGLIA

(a) *Protoplasmic Neuroglia.* In the middle and deeper layers of the cerebral cortex, and in the molecular layer of the cerebellum, are numerous cells which contain no fibrillæ stainable by the method of Weigert, but by Golgi's method revealing a wealth of fat processes. They were called by Andriezen protoplasmic neuroglia. Achúcarro was the first, after Golgi, to impregnate them successfully. They

may be easily demonstrated by the following methods, given in order of their usefulness:

In all these methods there are certain general principles to be remembered. In the first place they are chemical reactions and demand pure reagents and chemically clean utensils. It is also wise to use distilled water which has been redistilled over potassium permanganate as advised by Da Fano. Distilled water is indispensable unless definite directions are given to the contrary. "Formalin" always means a *neutral* 40 per cent solution of formaldehyde in distilled water. If it cannot be secured neutral, it must be neutralized by suspending in it powdered chalk, and filtered before using. Toning is always done with a yellow variety of gold chloride. Manipulation of the sections should be done with glass rods. It is necessary that the tissue be fresh, not over six hours post mortem in most cases, better one to two hours. Thin slabs must be placed in the fixative in order that it may penetrate rapidly. The length of fixation is often of the utmost importance and must be strictly observed.

I. *Gold Sublimate Method of Cajal* (10, 12).

1. Fix thin slabs of fresh tissue (not more than 6 hours post mortem) for 3 to 10 days in:

Formalin (neutralized with chalk), 15 c.cm.

Aq. dest., 85 c.cm.

Ammonium bromide, 2 gms.

2. Cut frozen sections 20–30 μ and receive in aq. dest. to which a few drops of formalin have been added.
3. After a rapid but thorough wash in aq. dest. to remove the formalin (2 or 3 changes) place in the following solution:

1 per cent aq. sol. of *brown* gold chloride, 6 c.cm.

Aq. dest., 35 c.cm.

Corrosive sublimate *crystals*, 0.5–0.8 gm.

Add the gold chloride to the water and dissolve the sublimate with the aid of heat. Filter while still warm. One must use crystallized sublimate and Merck's *brown* acid variety of gold chloride. Each 35 c.cm. of fluid will impregnate not more than half a dozen sections. Flatten the sections on the bottom of the vessel and place in the dark.

4. After 4–6 hours (at temperature of 20°–22° C.) the sections will be purple. They are then washed in abundant aq. dest. for a few minutes (5–10) and passed into the fixing bath:

5 per cent hyposulphite of soda, 40 c.cm.

95 per cent alcohol, 10 c.cm.

They are left here 6–10 minutes.

5. Wash for a few minutes in aq. dest. containing 30–40 per cent of alcohol, mount on the slide and blot with filter paper; abs. alc.; organum oil; xylol; xylol-Balsam.

The reaction is specific, impregnating intensely only the protoplasmic and fibrous neuroglia. If the time of action of the fixative

is prolonged beyond 20–30 days only the fibrous neuroglia are impregnated. The temperature of the gold bath is very important: for the cerebral cortex it should be 20°–22° C.; for the cerebellum, brain stem and spinal cord, 22°–26° C. For lower mammals a temperature of 24°–26° for 2–3 hours is necessary, and for reptiles, birds, fish and frogs, 25°–30° C.

The origin of the method lies in the gold sublimate procedure of Ziehen. The sublimate acts both as an accelerator and mordant. The use of bromide as an aid to glia staining was first proposed by Achúcarro.

II. *Silver Carbonate Method of del Río-Hortega (32).*

1. Fix in formalin-bromide (method 1,1) for 15–30 days.
2. Cut frozen sections 15–20 μ . Wash them in 2 or 3 changes of aq. dest. and place them, 3 or 4 sections to each 10 c.cm., in a solution of silver carbonate prepared in the following way:

Take 10 c.cm. of 10 per cent silver nitrate solution and add an equal or larger volume of a sat. sol. of lithium carbonate. Decant the supernatant liquid. Wash the ppt. with aq. dest. 50 c.cm. Decant again. Dilute strong ammonia with an equal volume of aq. dest. and add it drop by drop to the ppt. until it is all dissolved. Complete to 50 c.cm. with aq. dest.

3. After about 5 minutes in the oven at 45°–50° C. they will become greyish-yellow. (They should be moved about during this time to secure even impregnation.) They are then washed in aq. dest. and placed in 20 per cent formalin (neutral).
4. After 1 or 2 minutes they are rinsed in aq. dest. and placed in a 1:500 gold chloride solution until they turn an even violet shade.
5. Wash in aq. dest. quickly and fix in a 5 per cent solution of hyposulphite of soda.
6. Wash well in aq. dest.; 70 per cent alc.; 95 per cent; clear in phenol 5 pts., xylol 45 pts., creosote 50 pts.; xylol; xylol-Balsam.

Care must be taken to avoid an excess of ammonia in the silver carbonate solution.

III. *Río-Hortega's Fourth Variant of Achúcarro's Tannic Silver Method (33).*

1. Fix for several days in 10 per cent formalin.
2. Cut frozen sections 15–20 μ . Wash well in aq. dest. to remove formalin.
3. Heat for 10 minutes at 45°–50° C. in the following solution:
 Tannic acid, 3 gms.
 Ammonium bromide, 1 gm.
 Aq. dest., 100 c.cm.
4. Before the solution cools wash the sections with aq. dest. 20 c.cm. to which 2 drops of ammonia have been added just until they regain their transparency and flexibility.

5. Pass at once into a dilute ammoniacal silver solution (1 c.cm. of Bielschowsky's silver solution to 10 c.cm. of aq. dest.) until they become dark yellow.
 6. Wash quickly in aq. dest. and reduce in 20 per cent (neutral) formalin.
 7. Wash in aq. dest. and tone in gold chloride (see Method II, 4, 5).
 8. Wash in aq. dest. ; 70 per cent, 95 per cent, abs. als., origanum oil, xylol, xylol-Balsam.
- The method is not specific.

The tannic acid used should be the *Acidum tannicum puriss, leviss.* of Merck. The silver solution may be made in the following way as recommended by Hortege: To 30 c.cm. of a 10 per cent solution of silver nitrate add 40 drops of a 40 per cent solution of sodium hydroxide. Wash the precipitate a dozen times with aq. dest. (Use at least a liter of water.) Add 50 c.cm. of aq. dest. to the washed precipitate and then ammonia drop by drop until it is dissolved. Take care to avoid an excess of ammonia. Complete to 150 c.cm. with aq. dest. and keep in a dark place in a brown glass bottle.

The silver solution becomes brown from the tannic acid. It is best to have three dishes of the solution and transfer the sections to a fresh dish as soon as the liquid becomes turbid.

These methods reveal, in the middle and deeper layers of the cerebral cortex and in the molecular layer of the cerebellum, cells of the type shown in Fig. 1, A. They may be roughly subdivided into (1) bulky cells rich in cytoplasm and having large processes, (2) smaller ones with fewer appendices and (3) satellite or perineuronal cells. The first two types were recognized by Held, Eisath, Fieandt, Alzheimer, etc. The third type has been described principally by Cajal.

The nuclei of these so-called protoplasmic neuroglia cells are rather large and oval, with many heavy chromatin granules just under the nuclear membrane, and with heavy linin cords.

In the cytoplasm of the larger cells may be distinguished an endoplasm and an ectoplasm. The ectoplasm is much more heavily stained and is continuous with the processes. The smaller cells seem to be composed wholly of ectoplasm.

By the gold-sublimate method the cytoplasm both of the cell-body and its processes is seen to contain numerous small vacuoles. By the uranoformol (see page 344) and tannic-silver methods one sees not vacuoles but granules, heavily impregnated. They were seen by Held and Alzheimer. Fieandt, Eisath and Nageotte studied them. Fieandt gave them the name of gliosomes. Nageotte (19) considers them to be mitochondria. Achúcarro investigated them in great detail. They

vary from oval or elliptical forms to bacillary rods, and seem to be in large part of the nature of mitochondria. Other granules are occasionally found, sometimes forming wart-like excrescences on the larger processes. Pigment, large lipoid masses, vacuoles, and many other inclusions have been described by Alzheimer and others.

The centrosome was seen by Benda and Held, and its presence was confirmed by Fieandt. Achúcarro studied it in great detail. It is demonstrated very clearly by the gold-sublimate method on formalin-methyl alcohol material. It is usually a rather heavy granule, surrounded by a clear halo, and lying at the base of the largest process, often the sucker process. Its presence enables one to distinguish at a glance a glia cell from the nerve cell or third element.

A reticular apparatus of Golgi can be demonstrated in these cells of various animals, but has not been found in the adult human.

Held, Fieandt and Hardesty speak of a syncytium formed by these cells. Such a syncytium is not demonstrated by the methods of Golgi or Cox, which impregnate isolated elements over a more or less circular area. The uranoformol and tannic-silver methods often impregnate elements in this circular form. The gold-sublimate shows a most complicated interlacing of the cell processes, but nothing resembling a syncytium. Post mortem autolysis certainly takes place in isolated cells, and Alzheimer insisted long ago that in pathological processes the neuroglia cells acted as individual units. We may safely say that if a syncytium, such as described by Hardesty and others, exists in the central nervous system it is not formed by the neuroglia.

Probably every cell has one or more large processes, which end in an expansion or foot on the wall of a small blood vessel. It is not certain whether they extend into the space of Virchow-Robin, perhaps not; Achúcarro was accustomed to speak of these processes as the suction apparatus. It was his belief that by its means the cells removed substances from the blood stream, and perhaps secreted something into it by the same pathway. Nageotte has also proposed that these cells produce an internal secretion. The nutritive function of the perivascular feet was first suggested by Golgi, and afterward supported by Bevan-Lewis. Lugaro believes that they remove toxic substances from the blood which might be injurious to the nerve cells.

Many of the cell processes end around neurones, either the cell body or the principal dendrites.

Twin cells are often found with flat surfaces adjacent to each

other, and the centrosomes at opposite poles. They have undoubtedly recently divided. It is important to recognize this power which adult neuroglia cells have of dividing amitotically.

Many varieties have been described by various authors (marginal cells of Held, descending uniradial cells of Martinotti, comet cells of Retzius, perivascular cells of Andriezen, satellites of Cajal).

No fibrils of Ranvier-Weigert are present, although they may develop in pathologic conditions.

(b) *Fibrous Neuroglia*. It is this type of neuroglia which has been longest known, since it bears the fibers of Ranvier (22). It is the typical astrocyte, the easiest of all to stain, and therefore the most familiar. The modern impregnation methods were initiated by Achúcarro's tannic-silver. The gold sublimate and silver carbonate methods will give beautiful pictures on material fixed a month or more in formalin-bromide. The following will also be found useful for their demonstration.

IV. *Oxide of Ammoniacal Silver Method of Cajal* (9, 13).

1. Fix fresh tissue from 15 days to a year in formal-bromide (see method I, 1).
2. Cut frozen sections 12–25 μ and receive in the same fixative.
3. Transfer sections directly to the following solution:
Formalin (neutral), 6 c.cm.
Ammonium bromide, 3 gms.
Aq. dest., 50 c.cm.
4. After 4–6 hours in the oven at 30°–38° C. or 8–12 hours at room temperature, pass the sections quickly through two changes of aq. dest. and place them in the following silver solution: To 10 c.cm. of 10 per cent silver nitrate add 12 drops of 40 per cent sodium hydroxide. Agitate. Wash the ppt. 6 or 7 times with aq. dest. Dilute with 60–70 c.cm. of aq. dest. *before* adding ammonia. Add ammonia to dissolve. Avoid excess by leaving a small quantity of ppt. undissolved. Of this solution take 5 c.cm. in each of several porcelain dishes, and add to each 10–15 c.cm. of aq. dest. and 4 or 5 drops of pure pyridine.
5. Heat slightly over flame until the color of tobacco (if heated too strongly a ppt. will form).
6. Wash rapidly in aq. dest. 3–5 seconds.
7. Reduce in 5 per cent formalin 2–3 minutes.
8. Wash in aq. dest., and tone as usual (see Method II, 4, 5).
9. 70 per cent alc., 95 per cent, abs. alc., origanum oil, xylol, xylol-Balsam. The silver formulae of Bielschowsky or del Río-Hortega may be used just as well, depending on what one happens to have ready.

This method is the culmination of a long series of attempts to modify the silver method of Bielschowsky so as to get an impregnation of the neuroglia (Perusini, Montesano, Achúcarro).

V. *Uranoformal Method of Cajal* (13).

1. Pieces of fresh tissue not more than 3 or 4 mm. in thickness are fixed for 48 hours in the following liquid:
Uranium nitrate, 1 gm.
Formalin (neutral), 15 c.cm.
Aq. dest., 85 c.cm.
2. Wash rapidly in aq. dest., place in 1.5 per cent silver nitrate solution at room temperature for 2 or more days.
3. Rinse a few seconds in aq. dest. and place in the following reducing solution:
Hydroquinone, 1 gm.
Formalin, 10 c.cm.
Sodium sulphite (about) 0.5 gm.
(Just enough to give yellow color)
Aq. dest, q.s. ad 100 c.cm.
4. After 24 hours wash quickly in aq. dest. and cut frozen sections 20–30 μ .
5. Tone, dehydrate, mount (see preceding methods).

The finished block may be imbedded in paraffin or celloidin if desired. The method has the disadvantage of not penetrating well, so only the superficial sections should be used. It has the advantage of staining sharply the gliosomes and is especially useful for demonstrating the perivascular feet.

VI. *Hortega's First Variant of Achúcarro's Method* (33).

1. Fix at least ten days in 10 per cent formalin.
2. Cut frozen sections 15–25 μ . Receive in aq. dest.
3. Treat with 3:100 solution of tannic acid (ac. tan. lev. puriss. Merck) for 5 minutes at 50°–55° C.
4. Wash in 20 c.cm. of aq. dest. (to which has been added 4 drops of ammonia) until they have recovered their flexibility and transparency.
5. Pass at once into a dish containing 10 c.cm. of aq. dest. and 1 c.cm. of ammoniacal silver solution (see method III, 5). Pass through three such dishes, changing as soon as the fluid becomes colored, until the sections are dark brownish-yellow.
6. Wash in aq. dest.
7. Submerge in a 1:500 gold chloride solution at 40°–45° C. for 20–30 min.
8. Wash quickly in aq. dest. and fix in 5 per cent sodium hyposulphite for 5 min.
9. Wash in abundant aq. dest. Dehydrate and mount in balsam as in previous methods.

The method gives very clear and sharp pictures and brings out the nuclei, centrosomes, and other cell granules very well. It is very useful for demonstrating neuroglia fibrillæ, but is not specific.

A glance at Fig. 1, B, will tell more than many paragraphs of description concerning the general structure of the fibrous neuroglia.

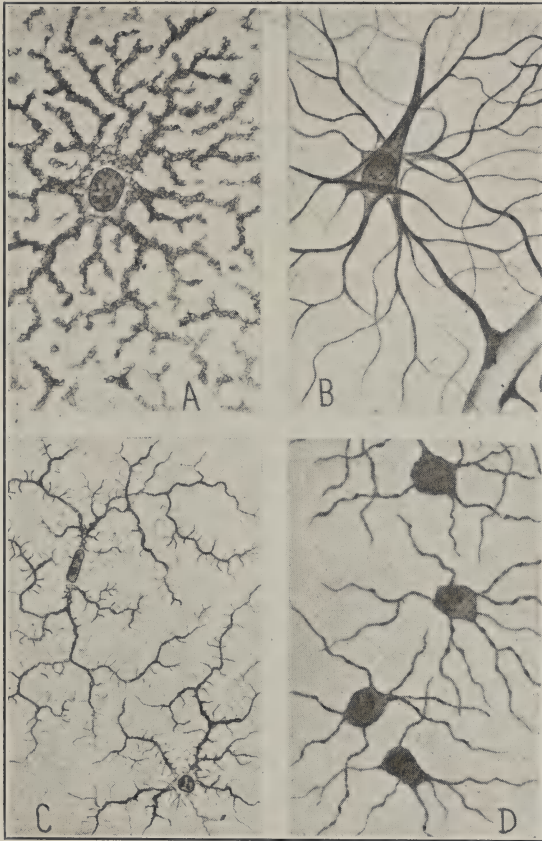


FIG. 1. The four types of interstitial cells. A—Protoplasmic neuroglia. B—Fibrous neuroglia. C—Microglia. D—Oligodendroglia. (After del Río-Hortega.)

This is the picture given by all the methods which have just been described.

The distinguishing feature of these cells is the presence of the fibrils of Ranvier (22). The new methods have definitely settled the old controversy concerning their nature. As is well known, Weigert considered them to be a true intercellular substance, completely emancipated from the cells. Cerletti believes they are artefacts.

Alzheimer, Eisath and Da Fano, among others, think that these fibrils develop inside the protoplasm of the cells and remain always attached to them. This latter view has been fully confirmed by the studies of Achúcarro (1), Cajal (12), and del Río-Hortega (27). The fibrils develop in the cytoplasm, beginning in the stoutest process, often the sucker foot, and invade gradually the entire cell. They remain always enveloped in protoplasm, at least in part.

The perivascular sucker feet are also a marked feature of these cells. They were well described by Golgi, Andriezen and others. The uranoformol method is particularly well adapted to their study and shows fibrillae in these processes which do not stop at the blood-vessel walls but continue on beyond into surrounding tissues. The feet usually stand on the vessels almost at a right angle.

It is interesting to note that the fibrous neuroglia cells have no gliosomes. There is also no Golgi reticular apparatus. The centrosome is a single heavy granule, usually at the base of the sucker process.

There are two types, (1) small cells with few processes and (2) large ones with numerous fibrillae.

MESOGLIA (?)

(a) *Microglia*. In 1918 del Río-Hortega succeeded, with his silver carbonate method, in impregnating clearly and specifically a homogeneous group of cells, which had previously been very resistive to all staining methods, and in this way settled many disputed points in the histopathologic structure of the central nervous system. These cells he called the *microglia*. Later he called them *mesoglia* to indicate his belief that they originated from the mesoderm. But Robertson had already applied the term *mesoglia* to a different type of cell, so it has seemed to us best to stick to the former name. In the beginning Hortega identified the microglia with Cajal's "third element," but there seems to be no doubt that the latter element corresponds to the "mesoglia" of Robertson (oligodendroglia of Hortega), and full credit may be given to Hortega for the establishment of a new histologic identity.

VII. Silver Carbonate Method of del Río-Hortega (29).

1. Pieces of fresh tissue not more than 3 or 4 mm. in thickness are fixed for 1-4 days, preferably 2 or 3, in formalin-bromide.
2. Heat the block in fresh fixative for 10 minutes at 50°-55° C. Cut sections by freezing method 20-30 μ . Receive sections in 25 c.cm. of aq. dest. to which 2 drops of ammonia have been added, for 10-20 minutes.

3. Wash rapidly in aq. dest.
4. Impregnate for 10 minutes at 15°–20° C. in the following solution:
 - 10 per cent sol. of nitrate of silver, 5 c.cm.
 - 5 per cent sol. of sodium carbonate, 20 c.cm.
 - Ammonia q.s. to dissolve ppt.
 - Aq. dest, 15 c.cm.
5. Wash in aq. dest. rapidly.
6. Reduce in 5:100 non-neutralized formalin for about one minute. Agitate the sections by blowing across the surface of the liquid. The sections will become darker.
7. Wash in aq. dest. Tone and fix as usual. Dehydrate. Clear in:
 - Phenol (crystal), 10 gms.
 - Beech creosote, 10 c.cm.
 - Xylol, 80 c.cm.
8. Wash in xylol and mount in Balsam.

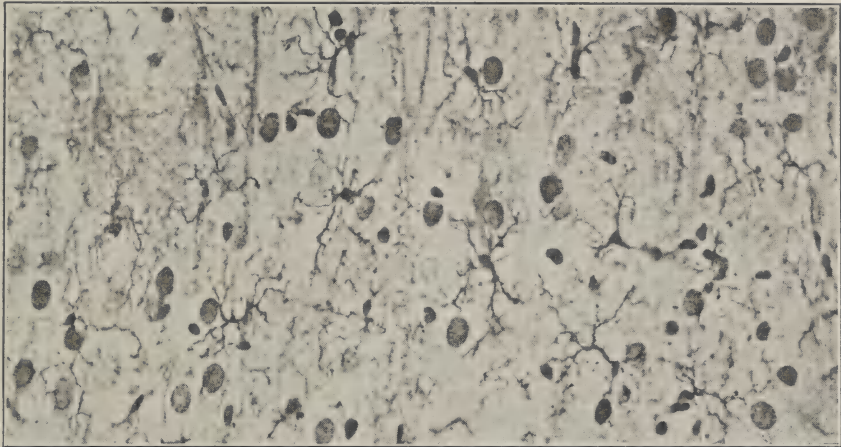


FIG. 2. Microphotograph showing microglia in cortex of rabbit. Hortege's method. (Personal preparation.) X-300.

The technique may be varied somewhat. After toning, the sections may be stained for fat by Herxheimer's method. The block may be cut and the sections heated in fresh fixative and this procedure sometimes gives a more complete impregnation, but does not seem to be so certain in its results. The accompanying microphotograph (Fig. 2) will give some idea of the average impregnation. All of these impregnation methods photograph badly because the sections are very thick.

The cells, as they are impregnated in this manner, are scattered all over the brain substance, but predominate in the gray matter. A typical example is depicted in Fig. 1, C.

The nucleus is ovoid or elongated, and has a heavy chromatin network. It is smaller than the nuclei of the other interstitial elements.

The cytoplasm is very scanty, and arising almost from the nucleus are 2, 3 or sometimes more processes, each of which divides dichotomously, often 2 or 3 times. The processes are covered with spiny excrescences coming off at right angles. By these spines they can be easily distinguished from the neuroglia cells. Occasionally lipid or pigment granules may be found, but there are no gliosomes, no glia fibrillae, no reticular apparatus and no centrosome.

The cells abound in the gray matter of the cerebrum, cerebellum and spinal cord where they are found as satellites of the neurones and blood vessels, and lying free in the tissues.

They are identical in structure in every vertebrate, in this respect differing clearly from the neuroglia cells. They are not fixed elements, but may migrate about in the tissues. Hortega was the first to give a clear description of them in the normal state, although the forms which they adopt when pathologically altered (rod cells, scavenger cells) have long been studied.

(b) *Oligodendroglia*. For a long time there has been recognized, in the vicinity of the neurones and along the long fiber tracts, round vesicular nuclei, surrounded by a clear area. All attempts failed to demonstrate processes coming from these nuclei. Cajal gave a clear description of them under the name of dwarf satellites or *third element*. Many investigators have wondered about them, and various names have been applied to them (nude nuclei, Shaper; indifferent cells, Bonome; rounded cells, Eisath; preameboid cells, Rosenthal; cuboid cells, Cerletti).

Bevan-Lewis, Bonome, Robertson, Nissl, Shaper and Rosenthal believed these cells to be undeveloped germinal glia cells. Held, Alzheimer, Fieandt, Jacob, Lugaro and Cajal were more cautious and said simply that by your present methods no processes could be demonstrated. Events have shown again how unwise it is to make negative statements concerning the microscopic structure of a cell. Alzheimer succeeded in staining the cytoplasm of these elements by his modification of Mallory's phosphotungstic acid-hematoxylin. Their cytoplasm is heavily impregnated by the uranoformol method of Cajal. Hortega was able to impregnate, at times, their processes by means of a modification of his silver carbonate. But certainly the most beautiful and complete demonstration of these cells is obtained by the platinum method of Robertson (25).

Robertson believed these cells to be of mesodermal origin and

called them "mesoglia." They must not be confused with Hortega's "mesoglia" (microglia). To avoid this confusion we have employed the terms *microglia* and *oligodendroglia*. It may well be that both are mesodermal elements.

VIII. *Hydrochloroplatinic Acid Method of Robertson (25)*

1. Fix pieces of tissue not over 3 mm. in thickness in 10 per cent formalin.
2. Transfer to a large volume of the following mixture and place in tightly corked bottle in the incubator at 37° C.:

20 per cent formalin (neutral)	}	—
1 per cent hydrochloroplatinic acid	}	aa

 Prepare freshly and filter before using. Deposition of platinum begins in 3–4 days. Tissues can be cut in 4–6 weeks. If the supply of platinum is exhausted the fluid loses its yellow color. In this case remove half the fluid and replace by 0.5 per cent solution of the salt.
3. Wash rapidly in aq. dest.
4. Place in watery dextrine solution 12–24 hours or longer.
5. Cut frozen sections 20 μ .
6. Clear, Dehydrate, mount as usual.

The dextrine solution is prepared as follows:

Dextrine, 140 grams.

Aq. dest., 280 grams.

Dissolve by boiling. Filter through cotton wool while still hot. When cool add 1 per cent of carbolic acid. For use add ammonia in the proportion of 10 drops of a 5 per cent solution to 30 c.cm. immediately before using.

The structure of the oligodendroglia, as shown by Robertson's method, may be seen in Fig. 1, D. By combining the results of the methods of Hortega, Robertson, and Cajal, (uranoformol), we may describe their structure as follows:

The nucleus is spherical and rather vesicular. Its size lies between that of the microglia and neuroglia nuclei.

The cytoplasm is unstained by ordinary methods, including Nissl's methylene blue. By the uranoformol method it appears homogeneous. Hortega's silver carbonate reveals, in many cases, a spongy or reticulated structure. There are small granules analogous to gliosomes, a rudimentary reticular apparatus, and a diplosome which is very difficult to stain.

The processes are slender, branch dichotomously but rarely, have no lateral excrescences, but show at irregular intervals small enlargements, in the center of which appears a vacuole by Hortega's method,

a granule by Robertson's. There are no fibrillæ of Ranvier, although the processes may sometimes appear fibrous, due to condensation of the protoplasm by reagents.

These cells are found, for the most part, in long rows along the long fiber tracts of the centrum ovale, bulb, protuberance and spinal cord. In the gray matter they are almost exclusively satellites, preferring the bases of the neurones. Sometimes they are found in the angle of branching capillaries (Cerletti's cuboid cells).

Robertson considered these cells to be of mesodermal origin and called them "mesoglia." Cajal also believes in a mesodermal origin and gives the following reasons: (1) they are not stained by his gold-sublimate method, which impregnates the finest ramifications of the neuroglia; (2) they appear late in embryonic development; (3) no regression of glioblasts to these dwarf forms has been observed; (4) they are never found, at any period of development, in the molecular layer of the cerebellum; (5) their number does not increase nor decrease in case of glial hypertrophy, and (6) Achúcarro, who studied them in senile dementia and other sclerotic diseases, could never find them forming fibrillæ of Ranvier.

Hortega believes them to be of ectodermal origin and gives the following arguments: (1) they are impregnated by the uranoformol method; (2) typical fibrous neuroglia cells are sometimes seen interspersed in a row of third elements along a fiber tract; and (3) the analogous subcapsular satellites of the ganglia are stained by Ehrlich's method, which never colors mesodermal elements.

The solution of the controversy will await a careful embryologic study. For the present Cajal seems to have the better of the argument. He thinks these cells are analogous to the Schwann sheath cells and the subcapsular satellites of the ganglia. It seems best at the present moment to employ the noncommittal term of *oligodendroglia*.

CONNECTIVE TISSUE

We have no intention of giving a description of the connective tissue of the brain. There can be no doubt, however, that the tannic-silver method of Achúcarro revealed the reticulum of the cortex with a clearness never before equaled, and has been very useful in pathologic investigation. For this reason an improvement of his method by Hortega is given. Hortega has also methods for collagen and elastin, but they seem to me to have no superiority over van Giesen's picro-fuchsin and Weigert's resorcin-fuchsin.

Two other impregnation methods are given, which we have found useful. Perdrau's method is the culmination of a long series of

attempts by Maresch, Snessarew, Tello, Achúcarro, and others, to modify Bielschowsky's ammonia-silver so as to obtain an impregnation of the connective tissue. Biondi's method is a clever variation of Cajal's gold-sublimate. Perdrau's procedure is based on the principle which Biondi first discovered, namely, treatment with permanganate and oxalic acid before impregnation.

IX. *Hortega's Second Variant of Achúcarro's Method* (33).

1. Fix in 10 per cent formalin.
2. Cut frozen sections 10–15 μ . Receive in aq. dest.
3. Treat for 5 minutes at 50°–55° C. or at 40°–45° C. for 15–30 minutes with a 1:100 solution of tannic acid in abs. alcohol.
4. Wash the sections in aq. dest., before they cool, just long enough to remove alcohol.
5. Place in ammoniacal silver solution at 1:10 in 2 or 3 small vessels, each containing 10 c.cm. (see method III). Leave until dark yellow.
6. Remove to aq. dest. and leave immovable on bottom of vessel until they become evenly darker.
7. Agitate slightly in aq. dest. and place in 20:100 formalin (neutral) for 1 minute.
8. Wash in aq. dest.
9. Tone, dehydrate, clear and mount as usual.

This method stains the reticulum of the connective tissue very sharply. Collagenic tissue is either unstained or much paler.

X. *Method of Biondi*.

1. Fix in formalin.
2. Cut sections 20–25 μ by freezing method.
3. Place 5–10 min. in a saturated solution of potassium permanganate (half sat. in summer). If the solution makes the sections too brittle to handle, stick them to the slide with blotting paper beforehand).
4. Wash briefly in aq. dest.
5. Bleach until white in:

Sat. sol. oxalic acid	}	—
Sat. sol. acid pot. sulphite	}	aa

Prepare just before using from 2 stock solutions and filter.
6. Wash well in aq. dest.
7. Immerse for 16–20 hours in:

1 per cent aq. sol. brown gold chloride,	10 c.cm.
5 per cent aq. sol. corrosive sublimate (crystal),	10 c.cm.
Aq. dest.,	50 c.cm.
8. Wash in several changes of aq. dest. for a few minutes.
9. 70 per cent, 95 per cent and abs. alc.; xylol; xylol-balsam.

These are the directions given by Biondi. One can make a combination of methods XI and I, following method XI up to (9) and

then method I from (3) onward. The impregnations are always granular, unsuited for high power work, and inferior to those obtained by IX and XI. The best preparation of the connective tissue on formalin-bromide material may be obtained by method IV.

XI. *Perdrau's Modification of the Bielschowsky Method* (21).

1. Fix in 10 per cent formalin.
2. Wash blocks 12-24 hours in running tap water, then in aq. dest., several changes, for 24 hours.
3. Cut frozen sections 15-25 μ , depending on tissues.
4. Leave in aq. dest. 24 hours.
5. Treat sections for 10 min. with 0.25 per cent pot. permanganate.
6. Wash in aq. dest.
7. Place in Pal's decolorizer until white:

1 per cent oxalic acid	}	—
1 per cent acid pot. sulphite	}	aa
8. Wash thoroughly in several changes of aq. dest. overnight.
9. Place in 2 per cent silver nitrate in the dark for 24 hours.
10. Wash in aq. dest. not longer than 5 min.
11. Treat sections 40-60 min. in Bielschowsky's silver solution prepared as follows:
Add 2 drops of 40 per cent sodium hydroxide to 5 c.cm. of 20 per cent silver nitrate. Just dissolve the resulting precipitate with ammonia. Dilute to 50 c.cm. and filter.
12. Wash quickly in aq. dest.
13. Reduce in 20 per cent formalin (not neutral) in tap water for 30 min.
14. Wash in aq. dest. Tone as usual with gold chloride.
15. Dehydrate, clear, and mount as usual.

By this method both reticulum and collagen are clearly stained reddish to black against an almost colorless field. The method is a little long but very constant in its results. It may be used on paraffin sections if the sections are not agitated in the solutions, otherwise they will fall off the slides. It is fairly specific, but may stain neuroglia fibrillæ. In this case prolong treatment with permanganate. Some idea of the results of this method may be obtained from the microphotograph, Fig. 3.

HISTOGENESIS

The earliest phases in the histogenesis of the central nervous system are not well understood. We know simply that in the epithelium of the neural tube are cells at rest and others in mitosis. The latter are called germinal cells (His). It is certain that the germinal cells give rise to neuroblasts. All stages of transition may be seen between them and apolar cells or primitive neuroblasts of Held (His). It is well established, also, that the indifferent, resting

epithelial cells may transform themselves into primitive spongioblasts, and that these latter may migrate to become spongioblasts or give rise by amitotic division to a spongioblast on the one hand and an ependymal cell on the other (Castro). We know, moreover, that the spongioblasts may divide amitotically, and that the neuroblasts do not divide, from the apolar stage onward. The transitions of neuro-

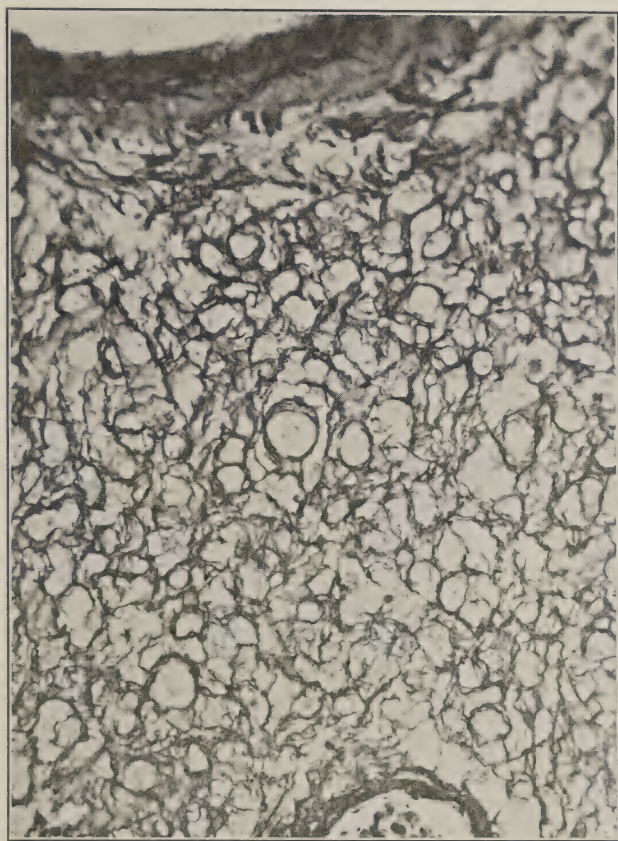
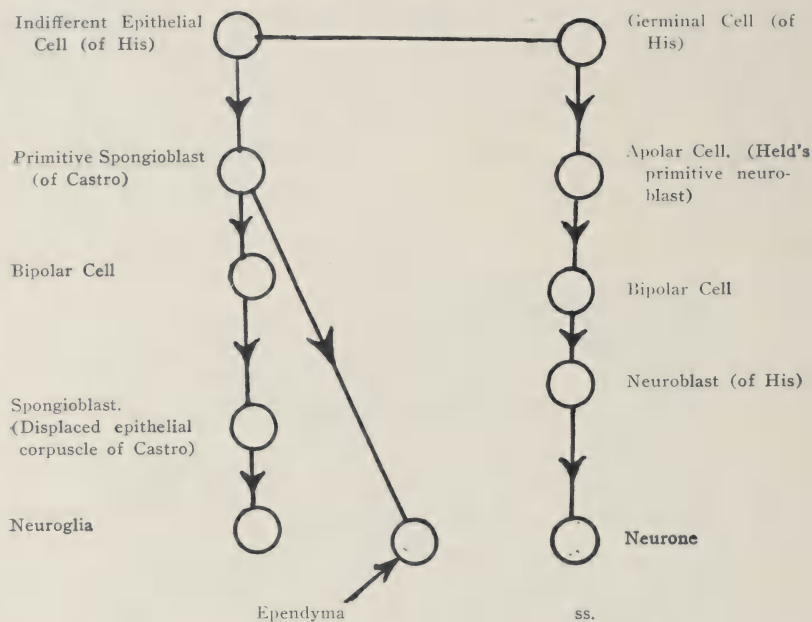


FIG. 3. Microphotograph of section of brain tumor. Perdrau's method. (Personal preparation.) X-300.

blasts into neurones and of spongioblasts into neuroglia have been followed in all stages. Transformation of spongioblasts into neurones has never been observed, but was an error of interpretation, due to the similarity of external appearance of spongioblasts and neuroblasts when impregnated by Golgi's method. The sum total of our actual knowledge may be represented by the following schema:



In the evolution of the neuroblast (Cajal, 14) the germinal cell of His is a globular cell with an ovoid nucleus, having a dense network of chromatin. The nucleus is often in mitosis. The cytoplasm is pale and finely granular. It cannot be impregnated with silver.

The primitive neuroblast of Held (apolar polygonal cell of Cajal) is a round cell with denser cytoplasm. The nucleus is vesicular but still has a rather heavy reticulum of chromatin. With Cajal's silver method may be demonstrated a neurofibrillary network in one pole of the cell, the fibrillogenous zone of Held.

The bipolar cell is spindle-shaped, the peripheral process being much coarser and longer than the central one. The nucleus has become still more vesicular and the neurofibrillary network extends into both processes.

The formation of the neuroblast of His is marked by the atrophy of the central process. The cell is now pear-shaped with heavy cytoplasm containing neurofibrillæ streaming out into the single peripheral process. The nucleus is ovoid with practically no chromatin, and one or two nucleoli.

The formation of dendrites marks the transformation into a neurone. Only after the dendrites have begun to form does the cytoplasm at the periphery begin to show an affinity for methylene

blue. The formation of the tigroid substance proceeds from the periphery toward the nucleus. The process begins in the human embryo of 20–30 mm. C.R. length. Myelin is formed very late, in the second half of uterine life. The nuclei are of two types: (1) in the granules of the cerebellum and the bipolar cells of the retina, the nuclei have a heavy reticulum with granules of chromatin; (2) the nuclei of all the other neurones have the chromatin condensed in one or two round solid nucleoli, otherwise there is only an extremely delicate linin reticulum. The centrosome early migrates to the periphery of the cytoplasm where it remains as a diplosome, demonstrable with the greatest difficulty (Hortega).

In the evolution of the spongioblast (Castro, 16) the indifferent epithelial cells are elongated columnar elements with the nuclei at various levels. The external half of the cells becomes reticulated.

The primitive spongioblasts are marked by the beginning of an affinity for gold-sublimate (Castro) and for Golgi's chromate. The ventricular ends are ciliated, with blepharoplasten in the form of diplosomes at the base of each cilium. The nuclei are round, with heavily reticulated chromatin network. They may be divided into two types, (1) those with the nucleus and the bulk of the cytoplasm near the ventricle, and (2) those with the nucleus and most of the cytoplasm away from the ventricle, but having a thick process going up to the ventricular surface. The latter have usually but one cilium and one diplosome. The internal extremities of these cells continue in long tails, which do not connect with the blood vessels. There is no syncytium.

The bipolar cell arises by amitotic division of the primitive spongioblast of the first type, with which it remains attached for some time by a long tail. They may arise from the second type also, which simply migrate away from the ventricular surface.

The spongioblast (dislocated corpuscle of Castro, primordial neuroglia cell of Cajal, astroblast of Lenhossek) is marked by the atrophy of the central extension. It is a pear-shaped cell, but in distinction from the neuroblast it has a nucleus with a heavy chromatin reticulum, a single heavy centrosome granule, and of course, no neurofibrillæ. Processes are put out from the end of the tail of the cells to the wall of blood-vessels. Later, processes go off from the side of the tails, nearer the cell body, to form sucker feet on the vessel walls in the neighborhood, and the rest of the tail beyond this point atrophies. Remnants of it may remain, however, in the fibrillæ, which, by the urano-formol method, may be seen to project beyond the sucker feet, over the vessel wall, into the tissues beyond. The

moment the cell loses its connections with the ventricular wall the centrosome shifts its position to the base of the largest perivascular process.

When the cell body begins to put out other ramifications the transformation into a neuroglia cell (astrocyte) is begun. They may be distinguished at once from neurones by the single heavy centrosome and the nucleus with numerous chromatin granules under the nuclear membrane. The fibrillæ of Ranvier develop in this phase, according

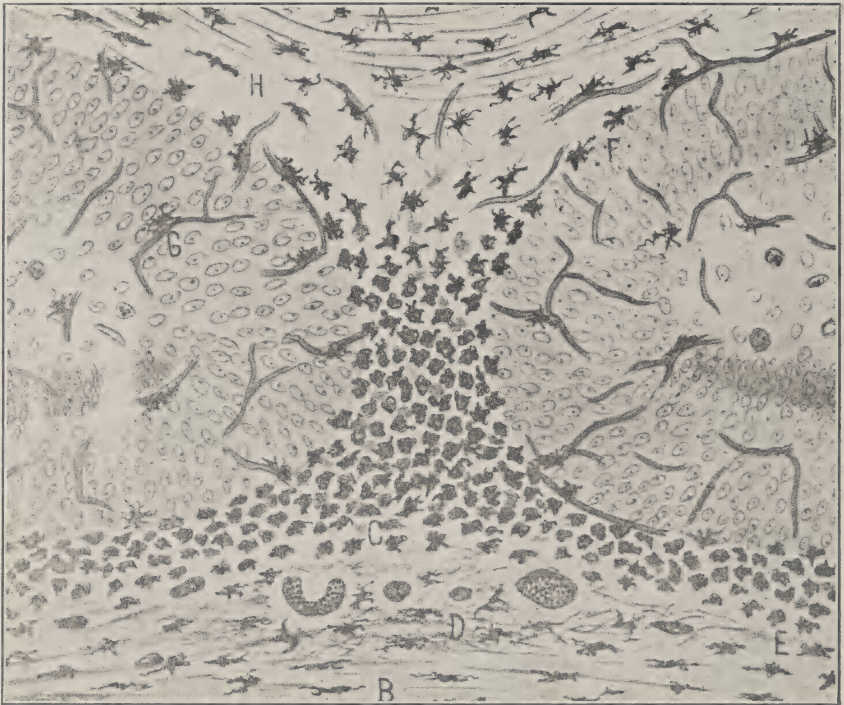


FIG. 4. Invasion of nervous system by microglia. Cerebrum of four-day rabbit. A—Corpus callosum. B—Velum. D, E, F, G, H—Microglia. (After del Río-Hortega.)

to Da Fano, about the fourth month of intrauterine life. Their development is practically complete at birth.

The finding of 2 or even 4 young astrocytes, poor in expansions, in groups, shows that, even in the adult, the neuroglia have the ability to divide. The “gliarassen” of Nissl are another evidence of this ability.

It will be seen that we have taken no notice of a possible mesodermal origin of the neuroglia. A pure ectodermal origin was pro-

posed by Boll (1874) and defended by Vignal. It was mainly established by Cajal in his studies on the chick embryo. If we understand *neuroglia* in the same strict sense as Cajal, this view is undoubtedly true.

But the belief has long been prevalent that mesodermic elements penetrate into the nervous tissue. Duval proposed it in 1877 and His insisted that it took place from the second month of intrauterine life. Capobianco, Hatai and Da Fano have also defended the same viewpoint.

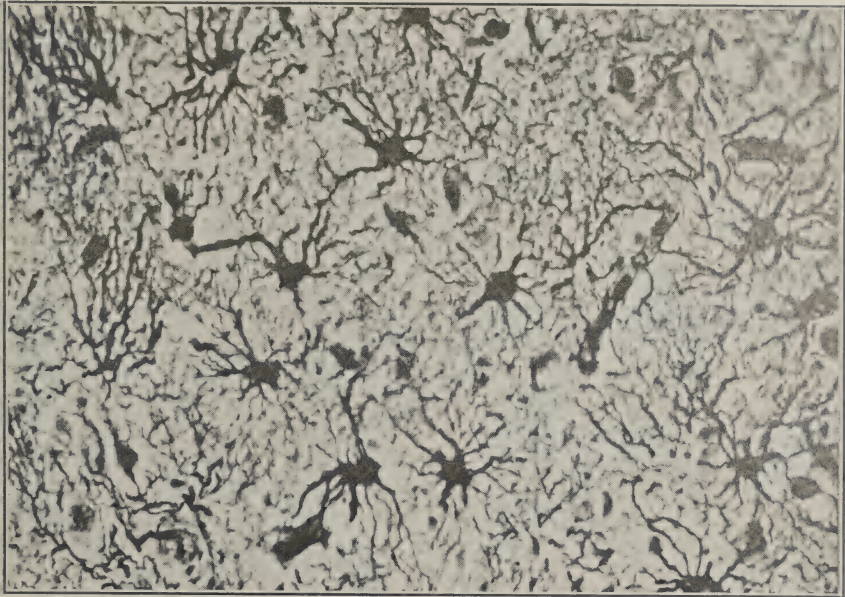


FIG. 5. Microphotograph showing cortical sclerosis near brain tumor. Cajal's gold-sublimate method. (Personal preparation.) X-300.

Del Río-Hortega has cleared up this point by his discovery of the microglia. He has shown by his careful embryologic work (29) how the microglia cells invade the nervous tissue from the pia mater, especially in the neighborhood of the inferior and superior choroidal telas, and spread out over the brain substance (see Fig. 4).

PATHOLOGIC ANATOMY

We have space for no more than a summary of the results which these methods have given in pathologic investigation.

a. *Scar Formation.* In the process of glia scar formation, it is interesting to note that the protoplasmic neuroglia cells may hyper-

trophy, lose their gliosomes and form fibrillæ of Ranvier-Weigert (Fig. 5).

b. *Amoeboid Cells*. Alzheimer (4) believed that in certain acute infections the neuroglia cells of the cortex became amoeboid and that this transformation was a progressive change (at least in part) destined to combat the infection. These amoeboid cells had abundant homogeneous cytoplasm, a small heavily stained nucleus, stubby processes and various granules and inclusions. In 1913 Achúcarro noted that these cells had all the marks of a regressive change.

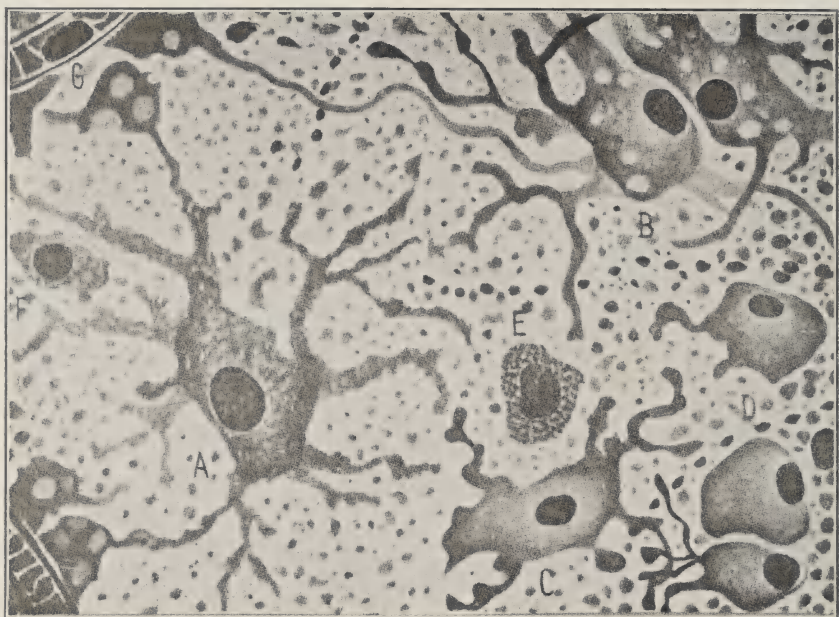


FIG. 6. Formation of amoeboid cells in subacute tuberculous meningo-encephalitis. (After Hortega.)

Almost at the same time Rosenthal showed that they might be produced by autolysis, and Buscaino that they might be produced experimentally by the action of alkaline and acid solutions. But the impregnation methods of Cajal (12) and Hortega (30) enabled all stages of the transformation of protoplasmic cells to amoeboid cells to be followed, and proved conclusively that they were always the result of a regressive change (Fig. 6).

Autolysis of the protoplasmic neuroglia cells may be well under way, in hot weather, within 3-4 hours post mortem, showing the necessity of securing fresh material.

c. *Rod Cells*. Since the investigations of Nissl (20) on general paralysis, certain elongated cells (stäbchenzellen) in the cerebral cortex have been well known to neuropathologists. They are characterized in Nissl's preparations by a long sausage-shaped nucleus from either pole of which extends a fat, straight process, palely stained, fading gradually into the background. They are almost always perpendicular to the surface of the cortex and parallel to the vessels. In their cytoplasm may be found various basophile, argento-phile, and sudanophile granules.



FIG. 7. Formation of scavenger cells from microglia 48 hours after cerebral trauma. (After Hortega.)

The origin and nature of these cells was for a long while disputed. Perusini, Straussler, Da Fano and others contended that they were of glial origin. Cerletti believed that they were of connective tissue origin, lying in fine connective bridges between capillaries. Achúcarro vacillated for a long time and finally concluded in 1914 with Gayarre (2) that they were mesodermal elements.

But it was left for Hortega (28) to clear up the controversy by

following all stages of their transformation from microglia cells. His silver carbonate method impregnates them distinctly and brings out the spiny excrescences so characteristic of the microglia.

d. *Scavenger Cells*. The phagocytic function of neuroglia cells was first proposed by Bevan-Lewis (6) who called them "scavenger cells." It is certain from his description that he was observing typical astrocytes. Marinesco called attention to the activity of certain cells near the dying neurones, and called the process "neuronophagia." The nature of these cells is uncertain, possibly both microglia and oligodendroglia. At any rate, there are well-known rounded cells to be seen in pathologic processes in the central nervous system, filled with lipoid droplets (possibly different from the Bevan-Lewis scavenger cells and from Marinesco's neuronophages) to which Merzbacher gave the name "abräumzellen." Many other names have been applied to them (gitterzellen, corps granuleux, fat granule bodies, etc.).

The origin and nature of these cells was long a matter of controversy. Some considered them to be of mesodermal origin (Nissl, Cajal, Stroebe, Ribbert, Bonome, Schmaus, Roccavilla, etc.), others of neuroglial origin (Schroeder, Jacob, Lotmar, etc.), while still others considered that they might have a double origin (Friedman, Merzbacher, Bonfiglio, Alzheimer, Lhermitte, etc.).

The possibility that some of them may be of vascular origin still remains. Ziveri (35), working with the gold-sublimate method, was unable to find any transformation of astrocytes into fat granule bodies in cases of cerebral softening, so that a neuroglial origin is unlikely.

The one fact definitely established is that they arise in vast numbers from the microglia. Hortega (31) has followed all stages of the transformation of microglia into fat granule bodies in the case of aseptic wounds of the cortex of the rat. The microglia cells accumulate from all the region around, swell up, thicken their processes, draw them in, fill up with fat and other debris and typical "gitterzellen" result (see Fig. 7).

The term scavenger cells seems the best that has been proposed, and may be retained as synonymous with "gitterzellen" and "corps granuleux," and must not be understood in the sense in which Bevan-Lewis first used it.

e. *Tumor formation*. In all inflammatory, hypertrophic and degenerative changes, the oligodendroglia remains strangely silent (possible exception in secondary degeneration in the spinal cord). They are not so inert in tumor formation. For the last two years we

have been investigating the structure and origin of so-called gliomata of the brain by means of these methods and hope soon to make known some of the interesting results which they have revealed.

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ANOSMIA, AGEUSIA AND REPRESSION NEUROSIS FOLLOWING BASAL FRACTURE OF THE SKULL

BY WILLIAM COLE, M.D.

JOHNSTON-WICKETT CLINIC, ANAHEIM, CALIFORNIA

While bilateral anosmia or unilateral ageusia are comparatively common following basal fractures of the skull, complete bilateral anosmia and ageusia due to basal fracture is rare. Undoubtedly this does occur following cerebral concussion from any cause, but in the majority of such cases it is either functional in character or transitory in its effects. The case presented herewith is of interest because evidently the loss of smell and taste functions are made up of both organic and functional components. It is also of interest because of the association with a typical repression or fear neurosis.

Case No. 310181. Lieutenant L. E. S. reported to my service in the Neurological clinic of the Department of Soldier's Civil Reestablishment at Winnipeg, Canada, on May 27, 1920, complaining of:

1. Loss of sense of taste and smell.
2. Tremor of the hands and body following excitement.
3. Being easily startled, excited or upset emotionally.
4. Dislikes talking about his flying experience.

Family history: Unimportant.

Personal history and present illness: Has never been sick in his life. Enlisted with Royal Air Force at Toronto, January, 1918. In May, 1918, he was assigned to the school of instruction in flying. In October, 1918, his plane crashed from a height of 1,000 feet. He was admitted to the Toronto General Hospital unconscious and bleeding from the nose, ears and mouth. Four incisor teeth had been knocked out. His nose had been flattened out and was corrected by operation. He said the diagnosis of his case was fracture of the base of the skull. He spent three months in hospital. Since the crash he has had absolute loss of the senses of taste and smell. He has also been very nervous and is easily startled or excited. His hands and body tremble on excitement. He states he has forgotten all about flying and the air force and hates to discuss his flying experiences.

Examination on Admission: General physical examination negative.

Examination of Nervous System: Gait and station normal. No incoördination, ataxia, asynergia or dysmetria. No adiadokokinesis. No astereognosis. Pupils round, equal and active. Fields normal. Ocular movements normal. No nystagmus nor strabismus. Hearing normal. Vestibular function normal. Sense of smell tested by Oleum Menth. Pip. Oleum Caryophylli. and Tr. Asafoetida. He is unable to recognize any of them and said he couldn't smell anything at all. Taste tested by powdered sugar, salt, dilute H.C.L. and quinine sulphate. None identified as sweet, bitter, salty or acid. The rest of the cranial nerves are normal. Grip strong and equal in each hand. Fine tremor of the extended fingers. Cutaneous reflexes present and lively. Knee kicks 2 plus on each side. Ankle jerks 2 plus on each side. Babinski and variants negative. No ankle or knee clonus. Biceps, triceps and supinator jerks plus one on each side. No disturbances of sensation or locomotion and no vasomotor or trophic disorders.

The only disturbances in the psychic field are exaggerated emotivity and complete repression of his flying experiences.

Treatment: The patient was admitted to the neurological hospital for treatment which consisted of rest, occupational therapy, physiotherapy and suitable recreation. He was encouraged to discuss his flying experiences with the other patients and not to spare himself on account of undue emotivity in doing so.

On the assumption that the anosmia and ageusia were functional in character, strong suggestion and the wire brush were tried out on several occasions. Although the patient did his best to coöperate no effect on the anosmia and ageusia was obtained.

Four weeks after admission the patient was discharged much improved and practically cured of his neurosis but the anosmia and ageusia remained.

Comment: The anosmia and ageusia in this patient's case are largely organic and due to injury to the olfactory bulbs, the nervous intermedius on both sides and to a lesser extent, the lingual nerve, chorda tympani and glossopharyngeal on each side. It is hard to believe that a patient could recover from a basal fracture sufficiently severe to interrupt all these nerves bilaterally. One would prefer to say there was a large admixture of organic and functional elements in the production of the anosmia and ageusia in this case.

A SECOND CASE OF ASCENDING COMPRESSION MYELITIS ASSOCIATED WITH UNUSUAL PATHOLOGY *

BY DR. HAROLD I. GOSLINE

PATHOLOGIST AND CLINICAL DIRECTOR

AND

DR. OWEN L. MURPHY

ASSISTANT PHYSICIAN, STATE HOSPITAL FOR MENTAL DISEASES, HOWARD, R. I.

The case which we wish to present at this time is related to the first case originally presented by Drs. Weatherby and Gosline in 1919 (*Vid JOURNAL NERVOUS AND MENTAL DISEASE*, 51:3, March, 1920, pp. 242-246) not only by reason of the fact that both were cases of ascending compression myelitis clinically, but because the unusual pathology found in the first case was found in this second case as well.

CLINICAL PRESENTATION (DR. MURPHY)

First Case. A review of the first case shows the following salient features: The patient, two days before admission to the hospital, fell backward during setting-up exercises, striking heavily on the ground. He felt pain in his left thigh at once and this spread up over his back. He walked but was somewhat stiff.

A few hours after admission to the hospital with a diagnosis of strained back, his left leg became paralyzed and on the next day the right leg, and there was retention of urine and feces.

That afternoon there was anesthesia over left upper abdomen to the level of the ninth thoracic nerve root. Diminished sensation and scattered areas of anesthesia existed on the right abdomen and involved the entire right leg. Anesthesia was most complete over the right thigh. Sensation in genital region was normal. Complete flaccid paralysis of both legs was found, together with absent abdominals, knee-jerks and ankle-jerks. Plantar response was slight flexion and cremasterics were hyperactive. There was pain or pressure over the spines of the eighth and ninth thoracic and violent paroxysmal pain in chest, bilaterally above the anesthetic area.

Two days later there was total anesthesia to touch, pain and temperature bilaterally, including genital region, to a line corresponding to the seventh thoracic nerve roots. Above was hyperesthesia, corresponding to fifth and sixth thoracic. Flaccid paralysis from waist

* Read at the meeting of the Boston Society of Psychiatry and Neurology, held on Thursday, April 19, 1923, at the Medical Library, 8 The Fenway, at 8:15 P.M.

down, reflexes absent except cremasteric which was now less active than before. The plantar response was flexion, then extension, then spasm of all muscles of the thigh for an instant, both sides acting similarly. Kernig not definite but spinal irritation evident. Severe pain in left shoulder and chest.

Second Case (H. L. No. 9591). An abstract of the second case follows: Family history is irrelevant. Past history not noteworthy till twenty (1910) when he had pneumonia followed by chronic bronchitis which he still has at twenty-six (1916). During his stay in the hospital he complained of pain in the chest, chronic cough, moderately productive and once raised some blood-tinged sputum. He often complained of weakness and malaise, had laryngitis, inflammation of the lachrymal sac. He was in the hospital seven years, when in March, 1923 (thirty-three) he developed influenza from which he appeared to be recovering, when he complained of urinary retention, constipation and pains in the lower dorsal and upper lumbar regions of a lancinating character. Later the same day he had a bilateral symmetrical hyperesthetic area over the entire lower part of the body below an elliptical line with concavity above, drawn between the anterior sixth costochondral junction and the seventh thoracic vertebra. Knee-jerks were sluggish, superficial reflexes absent. The following day he had to be catheterized. Abdominal distention developed together with bilateral flaccid paralysis. Heat, cold, pain and touch were absent and there was a belt-like area of hyperesthesia about five inches wide above the anesthetic area. Priapism was constant, together with spermatorrhea. Two days later there was ptosis of right upper lid and thick speech. Sacral decubitus developed. Pain disappeared, tissues were emaciated, eye grounds anemic, polyuria developed and finally a protracted and intractable hiccup. Before death, there was positive bilateral Kernig.

So much for the comparison of the neurological features of the two cases. Other clinical features were as follows: In Case I, on the second day after admission to the hospital there were signs of acute pleurisy on the right. X-ray showed no evidence of spinal lesion. There was no rise in temperature till the second day when there was an irregular elevation of from 1° to 3° F. Later, there was a septic temperature reaching 108° F. by axilla and death was due to respiratory failure.

In Case II, blood pressure was 110/70 on the second day. Two days later it was 100/72 and pulse had risen to 120. Blood pressure dropped to 92/72 shortly before death. Temperature varied irregularly from normal to 103.2° F.

A comparison of the laboratory tests will close this part of the paper. In Case I, lumbar puncture done on the second day was char-

acteristic of spinal cord compression and Wassermann was negative. The following day puncture showed Wassermann and gold sol negative, globulin slightly increased. A blood count showed 17,000 whites of which 72 per cent were polys and 28 per cent lymphocytes.

In Case II, the spinal fluid developed a fibrin clot in ten minutes, xanthochromia was present, only 3 c.c. of fluid could be obtained. Chemical tests were + + + +, the albumin solidifying with one drop of nitric acid. Cell count was impossible and the curve was 323333325. This curve was not of the usual type, but in the first tubes a clot formed which enmeshed the colloidal gold particles and completely cleared the fluid without changing the color of the gold particles from their original red. Wassermann on blood and spinal fluid were negative. The catheterized urine specimen showed an s.p.t. of albumin. The white count was 22,400, polys 71 per cent, monos, 24 per cent, transitionals 5 per cent.

On the mental side no notes were given in the first case but the individual seems to have been normal. He was a sergeant in the U. S. Army. The second case was one of dementia precox, paranoid. In the hospital he had various symptoms of a neurasthenic type and during his final illness he was restless and shouted out in pain so that he could be heard all over the ward. He gradually became sleepy, dull, apathetic, later muttering, from which he could be aroused only with difficulty.

PRESENTATION OF PATHOLOGY (DR. GOSLINE)

First Case. The essential features of the autopsy in the first case are as follows: The right pleural cavity contained clear yellow fluid, the left cloudy yellow fluid and was blocked off by extensive, loose adhesions. The right lung weighed 725 grams, the left 575 (with exudate). The right lung was purple mixed with dark red, the left lower lobe was covered by a dense yellow slightly adherent membrane and a small portion of this lobe sank in water. The cut section also showed a dark red area from which no air could be expressed. The mucous membrane of the upper respiratory tract was red and the tracheo-bronchial glands were markedly enlarged. The pericardial cavity contained cloudy yellow fluid. The heart muscle was softer than usual and the cut section showed dark red areas. Abdominal walls were flabby and sunken. Peritoneum was injected throughout and mesenteric glands were very much enlarged. The spleen showed an extensive distribution of fine red dots. The pia was injected throughout. The brain weighed 1,505 grams, was softened in focal areas. The choroid was reddish purple. On opening the spinal canal a large amount of thick, yellow creamy pus was found between the dura and the canal wall. The pia was injected. The cord felt

solid with the exception of the lumbar region which was very soft and on cutting the edge rolled out. In the thoracic region there was a communication between the spinal cord and the thorax on the left between the ribs. The microscopic examination of the cord showed complete loss of nerve cells with hemorrhages in all parts.

Second Case (H. L. 1923. 17-184). The essential features of the autopsy in the second case are as follows: The right pupil was larger than the left and the sclera showed through the cornea. Over the right thoracic region were numerous discrete and confluent reddish blebs. In the right pleural cavity was about 100 c.c. of cloudy reddish yellow fluid of moderately thick consistency. Over the left lung externally and posteriorly and especially over the lower lobe was a film of yellowish exudate and this side contained a small quantity of reddish opaque fluid of thin consistency. The right lung weighed 685, the left 680 grams. The right lung was purplish red for the most part and in the membrane, which was adherent to the lung were deep red areas. Sections of the lung beneath the membrane had collapsed. No air could be expressed and they sank in water. Mucous membrane of the respiratory tract was injected. The pericardial cavity contained reddish, fairly transparent, fluid. The heart muscle was very soft. The cut section was red. The large intestine was tremendously distended and the transverse colon formed a loop, the lower margin of which was midway between the umbilicus and pubis. The bladder was adherent to the anterior abdominal wall and posteriorly to the sigmoid. The organs in the pelvis were injected. Spleen showed extensive distribution of fine pin-head-sized irregular dark reddish dots throughout. Normal markings were absent. Consistency was mushy. Section of the intestine showed nothing of note except areas of injection. The kidneys were dotted with numerous large and small yellowish dots, the largest being the size of a small pea. These dots were distributed in certain areas and not evenly throughout the kidney. The cut section showed a similar localized dissemination and the cut edge rolled out. The bladder mucous membrane showed numerous depressed areas with irregular edges and bases undermining the mucosa. These were discrete and confluent and the fluid in the bladder was yellowish, opaque, tinged with red. The pia over the brain was injected and along some of the fine vessels of the vertex there was a whitish and, in others, a yellowish hue. The choroid of the ventricles had a similar color. Brain weighed 1,450 grams. The whole brain was almost mushy and section of the cerebellum and medulla showed nothing except softening. On cutting through the laminae in the right lumbar region there was a small gush of pus. Opening into the spinal canal the dura was covered with thick creamy yellowish pus with a slight greenish tinge from the second thoracic vertebra to the mid-lumbar region. The spinal cord in the lower lumbar region showed normal differentiation and consistency was slightly diminished. From the upper lumbar to mid-thoracic region cord substance was almost confluent and differentiation between gray and white was absent,

the cord substance resembling whitish pus in consistency and appearance. Smear showed large Gram positive diplococci, probably pneumococci.

The chief point of interest in these two cases is the hemorrhagic nature of certain parts and the purulent nature of others.

In the first case there was a hemorrhagic area in the left lower lobe, the heart muscle showed dark red areas, the spleen showed petechial hemorrhages, the microscopic section of the spinal cord showed hemorrhages. The second case showed hemorrhagic herpes, hemorrhagic pleural fluid, hemorrhages in the membrane covering the right lung, hemorrhages in the lung, in the heart muscle, petechial hemorrhages in the spleen.

As to the purulent nature of parts of the two cases, we have the following comparison: Case I showed cloudy fluid in the left pleural cavity, a fibrino-purulent membrane over the left lower lobe, cloudy pericardial fluid, purulent extradural exudate. Case II showed cloudy fluid in both pleural cavities, fibrino-purulent exudate over the left lung, multiple abscesses in the kidneys, multiple ulcers in the bladder, purulent exudate along some vessels of the pia, fibrino-purulent exudate of a greenish tinge extradurally in the spinal canal, due to probable pneumococcus.

The second case was one of influenza, occurring during the endemic outbreak of this late winter (1923) in this hospital. The first case was probably one of influenza occurring at the end of the pandemic of 1918-1919, though the diagnosis "influenza" was submerged in the other findings.

The presence of influenza, of the hemorrhages and of the purulent exudates follows very closely what is known both clinically and experimentally about influenza. I refer especially to the work of recent years done at the Rockefeller Institute for Medical Research (Vid. *J. Expt. Med.*, XXXIII, 2, 3, 6; XXXIV, 1). According to that work, the causative organism, bacterium, pneumosintes, injures the lung tissue allowing other ordinary inhabitants to invade the injured tissue. This conception of a disease process promises to open up extensive new fields of research. The same conception has long been held by some for tubercle and syphilis, though not perhaps expressed in so many words.

As to the second case, which was one of mental disease, it seems a proper idea to introduce for consideration here whether this patient may not have been a carrier. His clinical history of chronic bronchitis from the age of twenty, when he had pneumonia, till the age of thirty-three, when he died, favors the assumption that he carried

a chronic focus of infection in his respiratory tract. His spleen showed signs of exhaustion and his blood count done before his acute illness showed some drawing on his lymphocytes as well as a reduction in reds and hemoglobin (Sept. 3, 1921—Reds 4,016,000, Whites 9,200, Hb. 65 per cent, Polys. 68 per cent, Monos. 27 per cent, Trans. 5 per cent). Even during his final illness the lymphocyte count was relatively high for any ordinary septic process. The same was true of the first case but the spleen showed no signs of exhaustion.

It seems to me from what we know of the general pathology of mental disease that we may conclude that a certain proportion of such disease is due to some low grade, chronic process. Secondly, much of this trouble affects and exhausts the hemolymph apparatus, as shown by the blood studies during life and by the splenic exhaustion, the most constant feature at the postmortem table in mental cases.

To explain the relationship between the physical disease and the mental symptom is another problem. However, a beginning has been made (*Am. J. Psychiatry*, II: 2, October, 1922, pp. 235-257). We know that disease agents produce signs of increased activity, increased irritability, increased sensitiveness in many cases. (Some produce depression of function from the start.) Later, these irritating agents produce depression of function, especially if they go beyond the stage of stimulation and injure or destroy tissue. Now following this cue, mental symptoms can be arranged into those of an hyperactive sort or of an hypoactive sort. The extreme case of hypoactivity, namely, paralysis of function, is also frequently seen in mental symptoms.

The case which we have before us (Case II) had signs of increased irritability, increased sensitiveness, increased activity from start to finish. His mental symptoms at twenty-three followed his physical disease at twenty and paralleled his physical disease from start to finish. We may look to the future for final proof of this relationship between the physical carrier state and mental disease, but I believe that it is an opportune time to publish the idea now and I believe that the observation is a relevant one.

At this point we wish to acknowledge the courtesy of Dr. Geo. B. Coon, Assistant Superintendent, State Hospital for Mental Diseases, Howard, R. I., on whose service the second case occurred, and of Dr. Arthur H. Harrington, Superintendent of this Hospital, for permission to publish our work.

EPIDEMIC (LETHARGIC) ENCEPHALITIS *

A CLINICAL STUDY OF THIRTY-FIVE CASES GATHERED DURING THE RECENT EPIDEMIC, INCLUDING SOME RETROSPECTIVE CASES

BY MAX H. WEINBERG, M.D.

ASSISTANT NEUROLOGIST, WESTERN PENNSYLVANIA HOSPITAL,
PITTSBURGH, PA.

(Continued from page 280)

Case 9. Radicular pains, generalized tremors, meningeal and cerebral edema symptoms: Rev. H. E.; age thirty-eight; minister; No. 7481. Admitted 3/31/20. Died 4/7/20. F. H.—Negative. P. H.—Typhoid fever eighteen years ago. Influenza(?) four weeks ago. Facial paralysis eight years ago. C. C.—Pain over the occiput and in the left side of the trunk. Soreness of the left arm.

The patient had influenza four weeks ago and says that he never fully recovered from it. Felt tired and worn out and could not sleep well.

Three nights ago the patient dreamed that somebody twisted his neck off. When he awoke in the morning, he had sharp pain in the occiput and down the back of the neck and around the side of the left subcostal region. The pains are intermittent. He has not slept well since the onset of the present illness. He has had soreness of the left forearm and hand, and of the right upper arm. The left hand is weak and the patient says that he has dropped articles held in the left hand.

4/1/20. Dullness over the lower left chest, more behind. The patient is delirious this morning. Weakness of the right face, tremor of the jaw, tremor of the hands, more in the right. The patient is confused and disoriented. The deep reflexes of arms are increased, more on right. Knee jerks are increased, more on right. Achilles increased on left, not elicited on right. Right upper abdominal reflex absent. Fibrillary movements of the muscles present.

4/3/20. The patient is confused. He feels anxious but does not know about what. Tremulousness still persists. Pupils contracted.

4/5/20. Arms, hands, and head tremulous. Right hand more than the left. Right upper lip drawn up slightly and twitching. Left face somewhat flattened out. Keeps right lid more closed than left. Dysphagia developed to-day. Slight rigidity of extremities, more on

*From the service of Dr. W. K. Walker.

left, and head is retracted and neck rigid. Dermographia well marked. Kernig present, more on right. Deep reflexes increased. Abdominal reflexes absent.

4/6/20. The patient is perspiring profusely. Muscular tremors much more pronounced than the day before. Rigidity of the neck pronounced. Pupils respond slightly to light. Left eye slightly open. Kernig more marked on right. The patient died on the 7th.

Temperature on admission 101, and went up gradually from time to time, reaching 105 ante-mortem. Pulse followed the temperature. On admission 100 and rose to 150 ante-mortem. Respiration on admission 30, and reached 50 ante-mortem.

Case 10. Pains in trigeminal distribution, diplopia, paresis of facial muscle and motor fifth, rash and desquamation, and mild lethargy: Miss N. C.; school girl; age seventeen; No. 7493. Admitted 4/1/20. Discharged 5/24/20. P. H.—Influenza one year ago. C. C.—Drowsiness.

The sickness started ten days before admission with sharp continuous pain in the left side of the face which lasted three days. Drowsiness gradually disappeared after subsidence of the pain. Diplopia was present during the first three days. Tinnitus has been present up until the last two days. Vertigo bothersome at onset. No history of illness preceding onset of present condition.

Neurological examination—The patient presents a blank apathetic appearance. Facial folds partly obliterated on right. Weakness of facial muscles on right. Weakness of right masseter. The right pupil reacts but little to light. Both pupils are dilated. No nystagmus or extra-ocular palsies. Tongue heavily coated. It appears to protrude toward left. Elbow and knee jerks not obtained. No weakness noted in the extremities. No tremor. No ankle clonus.

Additional developments during the illness were, an extreme sluggishness of reaction to light, and dilatation of the left pupil, paralysis of the left face, paresis of the right external rectus, increased knee-jerks and left achilles, loss of right achilles reflex, erythema of face, and desquamation over abdomen.

During the first week, the patient did not see clearly what was going on about her, and later had but a vague recollection of her first week's stay in the hospital. She had no anxiety during that period. Temperature was 99 on admission, and rose to 100, and after a few days, normal, except for two days during the fifth week when it rose to 101 owing to a slight cold, and subsided in two days. Pulse between 80 and 90. Respiration normal.

Reëxamination. 4/28/22. She complains of pain above left eye. This comes on in spells, and lasts about two or three hours. Frequency about two months. Everything gets black before her. Once in July 1921, she fell in a faint. Occasionally she feels pain in the left leg. She feels weak all the time. Sight is still poor at times.

She is very irritable occasionally. Memory is poor. No emotional instability. Keeps company with young men and participates in

all the young people's activities. At times she gets very nervous. For instance, in the morning of this examination, when she was on an escalator in one of the department stores, she had marked palpitation and she commenced to cry. She is afraid of high places.

Neurologically she presented the following: Suggestion of masked face. Both pupils dilated. Left reacts faintly to light, right is sluggish to light; all other pupillary reactions normal. There is a slight weakness of the left external rectus. Tongue is in mid-line, restless, and is somewhat atrophied on left. Slight weakness of left face. No tremor of hands. Grip is good. Arm reflexes normal. Knee jerks, especially left, increased. Both achilles very much increased—left, almost clonic in response. No Babinski. No sensory disturbances.

Case 11. Convulsions, paralysis, conjugate deviation, contractures, meningeal symptoms, juvenile Parkinsonian syndrome, and pituitary insufficiency: W. P.; age seven; No. 7541. Admitted 3/31/20. Discharged 7/14/20. F. H.—Negative. C. C.—He was brought into the hospital by his father who states that he fell and “knocked his eyes crooked.” He does not know whether or not the patient was unconscious at the time. The father states that after this injury the patient became dull and listless, whereas before, he was bright and alert all the time. Has not been able to walk since injury. No more details of injury obtainable.

Neurological examination—The patient is in semi-comatose condition. Responds very slowly and indistinctly to questions. The eyes show nystagmus to left. The eyes are turned upward and to the left. They cannot be moved beyond mid-line to right. Pupils regular, equal, and react to light and accommodation. Right face paralyzed. Tongue protrudes in mid-line. All reflexes seem to be normal. No areas of anesthesia. Station cannot be determined owing to inability to stand up and his tendency to fall to the left. He is unable to walk.

4/4/20. The patient is in stuporous condition. Eyes turned upward and outward. Right face paralyzed central type. Nystagmus to left. Neck rigid. Right elbow jerk increased, left normal. Left knee jerk apparently increased, right diminished. No Babinski, or ankle clonus. Opisthatonos and tache cerebrales present.

Eye examination by Dr. Carson shows negative fundi. Only edges of discs could be seen owing to the eyes being turned up.

The patient ran a protracted and severe course. In about a week he developed diplopia, paralysis of the left external rectus, marked emaciation, scaphoid abdomen, contractures of lower limbs in flexion, more marked on left. This kept up for about four weeks. Then he developed marked tremulousness all over the body. The hands, very much wrinkled, were held in the typical Parkinsonian position and showed the tremor characteristic of this disease. The hands looked like that of an old man. The patient seemed to suffer extreme pain. About ten days after, he developed a Babinski on the

left, and the emaciation became extreme. He was unable to take nourishment. A few days later he developed a macular rash which disappeared in 24 hours.

The picture was fairly constant for about four weeks. He suddenly commenced to gain weight, and enough food could not be supplied. He gained so much in eight days the markedly emaciated patient became a chubby little boy. He had a masked face. Tremors of the hands were pronounced and he held them in the typical Parkinsonian position.

7/10/20. The following note was made prior to his discharge and over three months after admission: Facial appearance mask-like. Face well filled out. He shows tendency to smile unduly. Muscles slightly rigid. Slight tremor. Tongue deviated to right and is tremulous. Both pupils react to light. Knee reflexes increased. Achilles very much increased. On walking, the patient holds himself very rigid. The left hand still has tendency to hold metacarpophalangeal joints flexed, and phalangeal joints extended.

The patient has a ravenous appetite, and besides his own meals, he eats all the left-overs of the other children on the ward.

Temperature on admission 100. Fluctuated between that and normal for two weeks. Then it rose to 101 for two days, and down to 99 for two and a half weeks, after which it was normal for two weeks, then he had another rise to 100 for two days, and then receded to normal. Pulse and respiration coincided with temperature. Never higher than 120 and 30 respectively.

Reëxamination. 12/5/22. The child is very stout. Both pupils are irregular and react poorly to light, especially left. Accommodation is poor. There is a marked nystagmus on lateral deviation to right and left, and slight vertical nystagmus. There is marked increase of vessels in the left fundus. The jaw deviates to the left. There is a definite weakness of left face. Tongue is very tremulous, but in mid-line. The mask face is extremely pronounced, and he has a typical Parkinsonian smile. There is marked hypotonia in the left wrist muscles, also slight atrophy of thenar muscles of the left hand. There is definite associated movement of the left foot whenever the patient moves the left hand—none on right. In this movement there is pointing of the toes downward, the heel is drawn up, and the knee flexes on the thigh to an angle of forty-five degrees. The deep reflexes of the arms are diminished. The knee jerks are increased, more on left. Both achilles are increased. No ankle clonus. Babinski suggestive on left—the great toe does not move on stimulation and there is fanning of the toes. Gordon positive on left. No Chaddock or Oppenheim. No Babinski or concomitant signs on right. Abdominal reflexes present. No sensory disturbance. There is slight cephaloptosis, and in addition the head is inclined slightly to the left, the chin pointing to the right. There is twitching of muscles of right eyelids—about thirty per minute. There is also a rapid spasmodic movement of the left trunk and head occurring about ten times per minute.

While there is no definite tremor of the outstretched hands, a definite Parkinsonian tremor can be brought out by having the patient turn his hands with fingers outstretched. There is pass pointing in the right hand, also adiadochokinesis. He finds it difficult to approximate fingers and thumb of the left hand.

The gait is very peculiar. The legs and feet, in walking, are thrown forward and the patient comes down on his heels first, the toes touching the ground afterwards. The left knee is held practically stiff when walking, although flexion of the knee is good. There is also a slight drag of the left leg on walking.

The patient is extremely bashful and slow in all his movements. He smiles a great deal in a silly manner. Talks very little and in monosyllables. He is quite unmanageable—according to the parents. He has a ravenous appetite. He is unable to learn anything in school and loafs the entire day around the police station of the town. He shows definite mental reduction.

Case 12. Mild lethargy, facial paralysis, muscular twitchings, atrophy, and confusion: R. A. L.; age forty-three; American; laborer; No. 7581. Admitted 4/5/20. Discharged 6/5/20. F. H.—Father and mother dead. Wife died of influenza. No children. No miscarriages by wife. P. H.—Measles, quinsy, and acute inflammatory rheumatism twenty years ago. Alcoholic paralysis two years ago. Habits—Heavy drinker up until two years ago. C. C.—Burning in the eyes and feet.

The patient was well up until two weeks ago. He got wet at this time and a friend gave him some Jamaica ginger, of which he drank a large amount. The next day he noticed a burning and cracking sensation in his arms and legs. He has been in bed most of the time in his room.

Neurological examination—There is ptosis of the left eyelid. Pupils equal. Very faint response to bright light. Reaction not maintained. Tongue protrudes to left. The right face is flattened, including all three branches. Marked twitching of nasolabial muscles on the left side. Hands broad. Slight atrophy of the interossei, and forearm muscles on right. No tenderness of peripheral nerves on pressure. The fingers of both hands are tremulous. Left elbow jerk and both knee jerks are absent. The right elbow jerk is diminished. No Babinski, or ankle clonus. Tremulousness of toes noted.

During the course of the disease, the patient showed periods of confusion, a change from absent reflexes to increased reflexes, occasional marked talkativeness, fibrillary twitchings of the muscles of the left arm, and lethargy of a few days duration. He also had difficult urination for a few days.

Temperature on admission 101 for three days, receded to 100 and lower for about a week, then had another rise to about 100, and stayed so for about a week, and then around normal. Pulse and respiration kept pace with temperature.

Case 13. Lethargy, confusion, euphoria, twitchings of left foot, pyramidal tract involvement, desquamation, and mild Parkinsonian syndrome. D. V.; age twenty-five; Serbian; laborer; single; No. 7665. Admitted 3/19/20. Discharged 5/25/20. F. H.—Father and mother dead. Cause unknown. One brother living and well. C. C.—About a week before admission, the disease started with a chill and vomiting. The vomiting kept up for three consecutive days. He has not slept since the onset of the disease. He complains of no pains. His mind wanders at times. The chief sentence which he repeats often is, "I want to go home." His right foot jerks often. He felt drowsy the day of the examination for the first time. At present he lies with eyes closed.

Neurological examination—The patient appears very sleepy and most of the time holds his lids partially closed. He responds readily to questions, but does not answer intelligently. He is incoherent and confused.

The pupils are unequal—right greater than left. They react to light sluggishly. Facial muscles, tongue, and hands are tremulous. Knee jerks unequal, right more active than left. Elbow jerks are normal. There is a constant twitching of the left foot.

About two weeks after admission he desquamated practically over the entire body. The deep reflexes were very inconstant. They became in turn increased and decreased. He also had an exhaustible ankle clonus. The pupils at one time were stationary to light but improved. He showed a Parkinsonian tremor of the left hand. He was rather euphoric all through the disease. He could not understand why he could not get up and be about, since he felt so good.

Temperature on admission 100.4, then normal in twelve hours. Occasionally there was a rise of half a degree above normal. Pulse between 96 and 70. Respiration normal.

Case 14. Lethargy, occupation delirium, marked pyramidal tract symptoms, third nerve paralysis, and bulbar symptoms: L. R.; age twenty-one; single; No. 8448. Admitted 5/8/20. Died 5/20/20. C. C.—Drowsiness.

The illness came on about a week before admission. Most of the time he was restless and delirious. Usually imagined himself at his work. He was very much confused.

Neurological examination—The patient is lethargic. Speech markedly slurred and very slow. He is confused.

The pupils are contracted and do not react to light. The eyes are restless. Slight weakness of both external recti. He protrudes tongue with difficulty and only partially. He has a masked face. Left face somewhat weak. Facial folds completely obliterated. All deep reflexes increased. Abdominal reflexes active.

Two days later, the left pupil was completely dilated and stationary to light, and he had marked dysarthria. Then he developed hypotonia of the left arm, and slight atrophy of the interossei of the right hand. The deep reflexes as well as the abdominal reflexes were

increased. Grip weaker on right. Babinski was positive on both sides, but more marked on right.

On the ninth day after admission, the patient became semi-comatose. He was unable to speak. His face was covered with heavy beads of perspiration. He made attempt to follow out commands. Both pupils were stationary. All the deep reflexes were increased. Babinski and Chaddock were positive. There was no ankle clonus. The patient died on the twelfth day after admission.

Temperature on admission 99, receded in twelve hours, and fluctuated during the first seven days between 97 and normal. On the ninth day it commenced to rise up to 101 and then there was a sudden ante-mortem rise to 106.5. Pulse at first fluctuated between 90 and 72, and during the last three days gradually rose to 130 ante-mortem. Respiration kept pace and ante-mortem was 36.

Case 15. Lethargy, euphoria, pyramidal tract symptoms, marked psychoneurosis: H. P.; age twenty-eight; single; grocer; No. 8592. Admitted 5/14/20. Discharged 7/9/20. C. C.—Pain in upper right chest.

About ten days before admission he began to feel out of sorts. He felt dizzy and nauseated, but did not vomit. He had high fever and was delirious, tossed about in bed and was very restless. He was delirious up to day before admission. Has had pains in the left arm. The pain usually begins in the fingers of the left hand and radiates upward.

Neurological examination—There is a masked face and facial tremor. Slight weakness of right face. Tongue deviated to right. The pupils are contracted, slightly irregular, and react to light and sympathetic, very sluggishly. The jaw is deviated to the right. The arm reflexes are increased, more on right. Both knee jerks are increased. Exhaustible ankle clonus on right. No Babinski.

In four days, the pupils were almost stationary to light, and the deep reflexes of the legs were markedly increased. There was an ankle clonus on left, and exhaustible ankle clonus on right. He was rather euphoric and had no insight, and insisted that he was well and on going home.

The neurological picture has not changed much, but in about three weeks he became extremely restless. He was afraid that he would not be able to stand it, and his appeal for relief was almost pathetic. He developed marked psychoneurosis.

Temperature on admission 99.4° F., receded for the first two days, then rose to 100.4, and then would vary between 99 and normal. Pulse at first 96, then between 86 and 70. Normal for almost three weeks, and then commenced to fluctuate with wide limits, between 120 and normal. Respiration fairly constantly normal.

The patient was readmitted to the hospital three weeks after his discharge with the complaint of dizziness, sleeplessness, and nervousness.

Reëxamination. 3/5/22. Complains of stomach trouble, dizziness, nervousness and restlessness.

Pupils somewhat irregular and react to light sluggishly. Accommodation normal. Consensual and sympathetic normal. Tongue deviated to right and tremulous. Slight weakness of right face. Pulse 76 sitting, 86 standing. No tremor of hands. Elbow jerks and radioperiosteals of both arms increased, more on right. Knee jerks and achilles increased, more on right. No Babinski. Abdominal reflexes markedly increased. Cremasteric reflexes are increased. No intention tremor. Psychic reactions the same as after discharge, markedly hysterical.

Case 16. Symptoms of general infection, restlessness, third nerve palsy, mild pyramidal tract involvement, and mild lethargy: H. C.; age twenty-seven; American; electrician; No. 8928. Admitted 5/29/20. Discharged 7/14/20. F. H.—Father and mother living and well. Two sisters and seven brothers living and well. Wife and one child living and well. P. H.—Usual disease of childhood. Pneumonia eight years ago. Habits—Good. C. C.—The patient was brought into the hospital by his parents on account of “cross eyes and flighty spells.”

The patient states that two weeks ago he suddenly developed some generalized pains in the abdomen. He had some nausea and vomiting, and was constipated for four days. The pain in the abdomen lasted almost four days. Then he developed pain in the head, frontal and temporal regions, and diplopia.

Almost five days after the onset of symptoms, his eyes became “bad.” He could not read or distinguish objects at any distance. About this time his parents noted that his eyes were “crossed.” The parents state that the patient has changed from a very quiet person to a very restless and talkative one during the last ten days.

Neurological examination—The patient is restless, talks rapidly, but halts frequently. He shifts about in bed constantly. He is drowsy. The pupils are contracted. The right is stationary, the left reacts sluggishly. Tremor of facial muscles. Speech somewhat slurred. Slight weakness of right face. Deep reflexes of arms normal. Right knee jerk normal. Left knee jerk and achilles are increased. There is a suggestion of Babinski and ankle clonus. Abdominal reflexes are present.

The picture had not changed much during his stay in the hospital at first. At the end of two weeks he commenced to improve, and left the hospital in fairly good condition.

Case 17. Radicular pains, third, sixth, and seventh nerve palsy, confusion, delirium, and marked major hysterical symptoms: Miss R. B.; age thirty-five; nurse; American. Admitted 1/14/20. Discharged 8/27/20. F. H.—Mother died of diabetes. Father living and well. Two brothers and one sister living and well. Four sisters and one brother died in infancy. P. H.—Usual childhood diseases.

Diphtheria. Operated on in 1911—appendix removed, and gall bladder drained. C. C.—Pain in upper right abdominal quadrant. Facial paralysis. Progressive blindness.

The illness started two weeks before with a chill, temperature, and soreness in the gall bladder region. She was nauseated and vomited. A diagnosis of cholecystitis was made. About three days ago she developed left facial paralysis. For the past three weeks the patient was troubled with progressive loss of vision. It started first with diplopia. Now she can hardly recognize objects.

At the beginning of the illness, she noticed that she was partially deaf, but this symptom has disappeared. She also had a terrific headache, chiefly at the base, and some in vault of head, but it cleared up. Also was very dizzy and had loud singing voices in her head. Both these symptoms have not cleared up completely. The patient's friend stated that she noticed her at times irrational and muttering to herself.

2/23/20. Pupils contracted. Very slight reaction to light. Elbow jerks slightly increased, more on right. Knee jerks active, more on right. Achilles absent. No Babinski. Complete left sided facial palsy. Tongue protruding in mid-line. Slight pitting of skin over tibials.

2/25/20. Eye examination by Dr. Carson. Fundi negative. Blurred vision complained of at distance, is due principally to the tendency to diplopia. There is also paresis of the muscles of accommodation in both eyes, but more marked on left side.

2/26/20. Notes by Dr. Wholey. During the examination it was brought out that the patient complained of certain hysterical symptoms, particularly of a feeling of bigness. Eight years ago and at the beginning of the present illness, she had experienced the same sensation.

For two weeks previous to the onset of the paralysis, she suffered from severe pains in her head. They were especially marked in the parieto-frontal region. During the first week, these pains did not interfere with her work, but then she became very dizzy and was put to bed. She then began to hear noises like "shifting engines"—"it was a fluttering." After searching in her room, she concluded the noise was in her head. She then developed the ear trouble, in which both ears seemed to tighten up, and the hearing was affected. For two days she had diplopia. The face became paralyzed at the end of the second week. She also had illusions. The flowered wall paper took on all sorts of forms, which grimaced, danced around and carried on.

At night she saw great processions of people in her room, but none ever spoke to her. She felt at times as if her head were detached from the body. She could see it at the foot of the bed and reached for it. She was afraid to go to sleep for fear of waking up crazy. She took one-quarter grain of morphine by mouth, which made her much worse; she did not rest until five o'clock in the morning. The processions were of nurses and people she had taught in hygiene

classes. She would feel that there were people around her. None ever spoke. Her hallucinatory experiences were increased at night.

Since coming to the hospital five days ago, she has had some of these experiences, but slept well.

2/28/20. Another eye examination by Dr. Carson gives the following conclusions: Fundi negative, paresis of left third nerve, paresis of sphincter iridis left and some of right, paresis of ciliary muscles in both eyes, more of left, moderate contraction of form fields.

3/16/20. The eye report shows that the patient no longer has diplopia, and that she can close the eyes well.

The facial paralysis and most of the organic symptoms cleared up about the early part of April. But then the patient developed an attack of hysteria major. She complained of complete blindness, severe photophobia and marked abdominal pains. For six weeks not a ray of light was permitted by her in the room. She had the nurses take various precautions to exclude light from the room. She refused to leave the bed, and made herself obnoxious to the nurses and the management. She then responded to powerful suggestion and after that improvement came rapidly.

Temperature about 99° F. first two weeks, after that normal. Pulse fluctuated more widely, especially during the latter part of her stay in the hospital. Respiration normal throughout.

A note received from this patient dated April 15, 1922, states, "Since last July I have had very little trouble with my eyes or stomach. But I get very tired and exhausted at times. I have not the endurance I had before I was sick."

Case 18. Radicular pains shifting from one part of body to the other, cranial nerve palsies, mild Parkinsonian and cerebellar syndromes, myoclonic movement, reversed sleep, and acute thyroiditis: Miss E. A.; age twenty; American; school teacher; No. 6531. Admitted 2/3/21. Discharged 3/25/21. F. H.—Negative. P. H.—Negative. Habits—Good. C. C.—Backache, dizziness, sore throat.

The patient has been sick for two weeks. She started with headache, dizziness, sore throat, diplopia, and slight increase in temperature, 99 plus. For the last ten days, she has had retropulsion, and general staggering. Headache occasionally very severe, mainly frontal. The patient says that she saw double only this morning and now complains of right eye being pulled to one side.

Neurological examination—The patient is slightly dull and apathetic. She answers questions slowly. The eyes are half shut. There is a suggestion of mask face. Both pupils slightly irregular. Left reacts to light unusually quickly—right slower. Consensual, sympathetic, and accommodation normal. There is weakness of the right external rectus. Suggestion of festinating movement of eyes, especially upward. Otherwise, ocular movements normal. Tongue in mid-line, slightly tremulous. Suggestive weakness of left face. No tremor of hands. Suggestion of muscular rigidity in left arm.

Thyroid much enlarged, especially isthmus. Suggestion of rigidity of neck. Tenderness to percussion in left temporal area. Elbow jerks diminished on left—increased on right. Radioperiosteals increased on both sides. Triceps increased on both sides. The abdomen is somewhat distended, reflexes not elicited. Knee jerks increased on both sides, especially left. Achilles increased on left, normal on right. Babinski, ankle clonus, and Oppenheim negative, but crossed reflex from the right to the left on testing for Oppenheim is noted. Suggestion of intention tremor. Adiadochokinesis on left. No dysmetria.

The patient is emotionally unstable, cries easily, and can give no reason for crying.

Four days later the patient complained of pain in the right side which radiated clear across to the left side. The deep reflexes were more increased, and the crossed Oppenheim reflex changed and was from left to right. Two days later she complained of numbness in the left arm and hand, and pain in the heels which was radiating from the knees down. She also had twitchings around the mouth.

Two days later she showed more active pupillary reactions, tremulous tongue with fibrillary contraction, and tremor of the hands, greater on the left. She complained of pain in the mastoid region. The next day she showed in addition a constant twitching of the right forearm. All previous symptoms and signs, however, were improved. Two days later she stated that she could sleep better in the day time than at night. Two days later I noted a sudden enlargement of the thyroid. Three days after that the thyroid became even larger, and the patient complained of pain along the left sternocleidomastoid, and hot flashes of pain across her lower back.

In a few days the patient cleared up fairly well, except that the right pupil still reacted somewhat sluggishly to light, and the knee jerks were increased, left more than right.

Temperature never above 99° F., and during the last four weeks normal. Pulse around 80, but rose sharply to 100–110 with the sudden enlargement of the thyroid for a day or so and receded to normal. About three weeks later, it again rose to 110, although the temperature was consistently normal. At the time the patient left the hospital the pulse was around 100. Respiration normal throughout, although for a while she complained of a cough.

Case 19. Radicular pains, severe myoclonic movements, reversed sleep, marked pyramidal tract involvement, and slight cranial nerve involvement: J. S.; age thirty-two; American; married; No. 6825. Admitted 2/14/21. Discharged 3/17/21. F. H.—Negative. P. H.—Negative. Four children. Wife had no miscarriages. C. C.—One week ago he had pain in the left shoulder radiating down the arm into the fingers along the course of the radial. Twitching of the left arm for the last three days. No history of a cold or respiratory system infection.

The patient was not stuporous until the doctor gave him a "hypodermic."

Neurological examination—The pupils are contracted. (Under M. S. probably.) Tongue deviated to the right. Deep reflexes of both arms much diminished. Right knee jerk diminished. Left knee jerk present. Achilles present. Babinski present on the right. Oppenheim, Chaddock, and Gordon negative.

Further examination impossible because of the stupor of the patient.

2/15/21. Masked face, labial fold completely obliterated on the right, partly obliterated on the left. Right pupil reacts to light sluggishly. Left pupil normal. Fundi show blurred margins. Tongue deviated to right. No evidence of facial palsy. Myoclonic twitching of left arm, about ten per minute. Deep reflexes on right diminished, on left normal. Continentia urea. Babinski on the right. No ankle clonus.

2/16/21. Jerks in the left arm, started again this morning and kept up ever since. Coarse tremor of outstretched hands. Pupils react sluggishly to light—left irregular. Speech changed, is monotonous. Babinski on right. Deep reflexes diminished. Patient is confused during the morning hours only.

Three days after admission he complained of inability to void. The twitchings of the left arm became diminished, but the tremor increased, and both external recti have become weak. Later he developed marked myoclonus of the right abdominal muscles.

The myoclonic movement of the right abdominal muscles was so marked that it pulled the head to the right side and at times shook the bed. The Babinski persisted positive on right, and Oppenheim positive on both sides.

In a few days the patient complained that his sleep was reversed. He slept all through the day but could not sleep at night. The twitchings of the right abdominal muscles were not so marked. The twitchings were at the rate of 16 per minute.

Both pupils became almost stationary to light, and the right arm was weaker than the left. About three weeks after admission, he complained of severe pain in both arms. The tongue was in mid-line.

3/3/21. Tremor of hands less marked. The facial paralysis cleared up. Babinski and Oppenheim marked on both sides. Desquamation of soles of the feet extending up to and around the toes very marked. All myoclonic phenomena disappeared.

Temperature between 101° F. and 99° F. during first three weeks, and then normal. Pulse kept with temperature, but later remained high. Respiration normal or slightly raised.

Case 20. Lethargy and stupor, mild hemiplegia, rotary nystagmus, bulbar symptoms, fifth cranial nerve palsies, and nephritic symptoms: C. M.; age thirty; laborer in steel mill; No. 7097. Admitted 2/24/21. Discharged 4/1/21. F. H. and P. H. could not be obtained. C. C.—Headache, double vision, difficulty in speaking, drowsiness, twitchings of the facial muscles.

The symptoms came on suddenly, three days before admission, when he became unconscious while at work. The mill physician diagnosed fracture of the skull.

2/27/21. Neurological examination—Pupils both contracted, right does not react to light, left has but a flicker of a reaction to light. Left face weak. Apparent weakness of the left external rectus. Protrudes tongue with difficulty. Tongue dark brown. Facial folds obliterated. Articulations difficult, due to labial paralysis. Grip of the right hand weak. Deep reflexes of arms are increased, more on the right. Abdominal reflexes diminished on right. Knee jerks—left normal, right increased. Achilles—left normal, right increased. Babinski, positive on both sides—more marked on right. Atrophy of right leg, five-eighths inch difference. Right leg slightly weak.

The patient is confused, disoriented, perseverates in motor field (told to raise leg, and then when asked to respond to sensory stimulus, continues to raise leg).

Seven days after admission he developed marked circular and rotary nystagmus. No rigidity of neck found. The tongue appeared very toxic. He was semi-stuporous, but gave evidence of being in marked pain.

3/5/21. Patient has mask face, mouth is half open. He looks depressed, is conscious, but prefers not to answer questions. Central rotary nystagmus is complete, right to left thirty in minute.

Pupils unequal, right greater than left, fairly regular. Right reacts fairly well to light, left not so well. Apparent paralysis right sixth nerve. Protrudes tongue with difficulty, and when urged to protrude, he merely opens mouth wider. Paralysis of right seventh nerve and motor portion of the right fifth marked. Deep reflexes of arms increased. Slight rigidity in the right arm. Abdominal reflexes on right practically absent, left normal. Knee jerks increased on the right, normal on left. Babinski marked on right. Exhaustible ankle clonus on right. Babinski present on left. No Gordon.

During the next two days the nystagmus cleared up completely. His speech was very thick. Within four days he developed tendency to undue laughter. He asked permission to get up and walk about. In the course of a few days, the paralysis cleared up and the only signs of bulbar involvement were the laughing and his hoarse, empty voice. The euphoria and disordered reflexes kept up until the time of his discharge. A slight lateral nystagmus developed on the last day of his stay in the hospital.

Temperature—Only once on the fourth day of admission up to 99° F., otherwise normal and subnormal. Pulse 80–90. Respiration normal.

Case 21. Diplopia, delirium, nystagmus, and mild pyramidal tract involvement: H. W.; age five; Caucasian; Jewish; No. 14990. Admitted 5/3/20. Discharged 6/6/20. C. C. —Insomnia, pain in posterior part of neck, in the right shoulder joint, and in fingers of left hand.

About nine days before the patient was brought to the hospital, he became suddenly ill. The very same night he complained of diplopia and was very restless. During that night he had a muttering delirium and carphylogy.

5/6/20. Neurological examination—Right pupil is slightly irregular. Reaction to light fair. There is lateral nystagmus to right, and vertical nystagmus on looking up. Tongue deviated to right, and heavily coated. Slight weakness of right face, probably central paralysis. Jaw reflex increased. Hypotonia of left hand. Grip weaker in left than right. Deep reflexes of right arm are increased, left normal. *Adiadochokinesis* on left. Ataxia in left arm. Abdominal reflexes present. Both knee jerks increased. Achilles absent on left, increased on right. No Babinski. Fanning of toes on taking Chaddock on left. Oppenheim positive on left. No ankle clonus.

Temperature was 100 on admission. During the first night it rose to 103, and after that stayed around 100 for about a week, after which it was about normal. Pulse between 100 and 120. Respiration between 36 and 48.

About three months after his discharge from the hospital, the patient had marked *cephaloptosis*, reversed sleep, and a few residual symptoms that he showed during the acute stage of the disease.

Reëxamination. 7/4/22. The boy is very much emaciated, chin sharp and pointed, facial folds, especially the right, very deep, and the general facial expression is that of an old man. The mother states that he is awfully nervous, is afraid to remain by himself, and has frequent nightmares. These nightmares are especially apt to occur when the boy is tired. According to the mother's statement, he is not the same boy he was before he took sick. When he does not feel well, or when he is especially tired, he is apt to sleep for a great length of time. Only a week before the examination, following a school picnic on Friday, the patient slept thirty-six hours straight before he could be awakened.

The pupils are unequal, dilated, left much more than right. Both react sluggishly to light, and sympathetic. Accommodation normal. There is definite twitching of right upper lid. Tongue very restless, tremulous, and deviated to right. Slight weakness of left face. There is no trace left of the *cephaloptosis*. Cervical glands enlarged on right. There is coarse tremor of the outstretched hands. All the deep reflexes are diminished. No Babinski or ankle clonus. The muscles of the arms and forearms are exceptionally wasted away. On walking, he has a slight drag of the right leg. No ataxia, no intention tremor, although the patient says that when he holds his books for any length of time his hands tremble.

Case 22. General infection, radicular pains, muscular atrophies, neuritis of right arm, desquamation, bulbar symptoms, coma, and decubitus: H. S.; age twenty-six; Roumanian Jew; married; No. 14877. Admitted 4/6/20. Discharged 6/6/20. C. C.—Pain in

the right shoulder joint and in the right elbow joint. Tenderness of the extensor muscles of the right forearm.

The patient became ill three days ago. In the evening he complained of pain in the right shoulder. The pain was very severe all of that night and since then has had acute pain. On the following day, the pain localized in the right elbow, and to-day the right forearm became tender. About two weeks ago, the patient had laryngitis and rhinitis. He never had an attack of pain like this before. The pain is continuous and sharp in character.

Difficult urination developed on the tenth day of the disease.

4/28/20. Both pupils are dilated, the right more than the left. React to light sluggishly. Left face weak. Protrudes tip of the tongue between teeth with difficulty. Speech barely audible. Some rigidity of the arms, also rigidity of the neck. Face is mask-like, patient seems to have a general twitching movement of all the muscles of the body, but it is not regular or rhythmical. Atrophy of the interossei muscles of both hands, and particularly of the thenar eminences. Arm reflexes fairly normal. Knee jerks both increased, left more so. There is bilateral ankle clonus, more marked on the right. Both achilles are increased, more marked on the right. Babinski on right. Slight Kernig on right. Cremasteric present. Suggestion of desquamation about both knees. The patient seems to be in pain.

During the sixth week of the disease, the patient became comatose. His temperature rose to 105.2. It looked like an ante-mortem rise, but the temperature dropped again the next day. A beginning decubitus was noted with the rise of the temperature, and as the pus commenced to discharge, he started on the upgrade and recovered shortly after.

The temperature was at first between 99° F. and 100° F. for the first four weeks, and then rose to 103 for a few days, and then to 105. After twenty-four hours, this came down to normal. Pulse and respiration kept pace with the temperature.

Reëxamination. 4/22/22. The patient complains of weakness and pain in the back, pain in right arm, especially when it rains. Is working at his trade, and is doing as well as he did before he took sick. Stayed away from work about a year. Sleeps well. No mental changes of any kind.

The patient presents a masked face. Both pupils are irregular, especially left. The left pupil reacts very poorly to light. The right pupil is but slightly better. All other reactions are slightly weakened. Ocular movements normal. Weakness of right face. Lower jaw deviates to right. Grip good. Arm reflexes normal. Right knee jerk normal. Left knee jerk and both achilles increased. He still protrudes tongue with difficulty. No sensory changes in right trigeminal area.

Case 23. Restlessness, mild delirium, rash, arthritic involvement, mask face, and third nerve involvement: J. S.; age sixteen; Amer-

ican; No. 14814. Admitted 3/20/20. Discharged 4/25/20. C. C.—Rash, restlessness, talkativeness, and general pains.

About a week before admission the patient complained of not feeling well. The next day he went to bed, was very restless and talkative, and could not sleep. This changed to lethargy when he was but a day in the hospital. There was a rash at both elbows, both knees, along the right axillary space, and on the big toe of the right foot.

3/21/20. The rash appeared on the day before. The patient is still restless. Pupils unequal, right greater than left. Right pupil irregular. There is but a flicker of reaction to light. Inflammatory condition of the big toe on the right side. Some of the other joints also show some inflammation, but not as marked. Commences to hold arms in the typical position—folded on abdomen.

He ran rather a mild course. The outstanding symptoms were a slightly masked face, and the mental symptoms mentioned above. The temperature never rose above 100.4° F. during the first few days, and after that was around normal. Pulse between 90 and 98 when the temperature was elevated, but after that was about normal. Respiration 30 during first few days, and then down to normal.

I had occasion to see this patient several times on the street. He promised to come down to be reexamined several times but always failed to keep his promise. He presents a mild Parkinsonian type, but I cannot give any details. According to his brother, a dentist, he changed in character very much. He refuses to go to school or work. He is irritable and forgetful. He cannot apply himself to any useful work.

Case 24. General infection, abdominal pains radiating in character, myoclonic movements, Parkinsonian syndrome, nocturnal delirium, cranial nerve involvement, muscular atrophies, bulbar symptoms, and coma: S. C.; age twenty-two; Russian Jew; single; grocer; No. 14577. Admitted 1/13/20. Died 2/23/20. C. C.—Constipation, pain in the left lower quadrant which radiates to the back. The pain is sharp and worse at night.

1/26/20. Neurological examination—Flush on the left side of face. The patient is muttering, and when he does talk, he talks very slowly. Clonic movement of back and hamstring muscles on both sides is present. The patient seems very weak. Talks with a nasal twang. Tongue very much coated. Biceps reflexes increased. Triceps diminished. Knee jerks and achilles lost. No Babinski or ankle clonus. Face greasy. Hands very tremulous on keeping them outstretched.

The patient was not seen by me until he was in the hospital two weeks. This was early, before the myoclonic type of encephalitis had been described, and I made the diagnosis of paramyoclonus multiplex from some underlying source of infection, because the only outstanding features were the myoclonic movements of the abdominal and hamstring muscles, and the marked asthenia.

Four days later, on finding slight changes in the pupillary reflexes, I realized that I was dealing with a new form of encephalitis. Two days later the patient complained of diplopia for the first time. There was hyperesthesia on inner border of legs, and slight hyperesthesia of trunk. The next day he developed a vertical and lateral nystagmus, a mask face, and a moderate rash on the body. The pupils then became stationary. Two days later he developed the complete picture of the disease, and the following note was made on that day:

2/7/20. Patient very drowsy, sleepy, talking with definite nasal twang. Protrudes tongue with difficulty. Mask-like face, greasy. Falls asleep between questions. Left pupil twice the size of the right. Both pupils react sluggishly to light, especially the left. Loss of both cremasteric reflexes. Hypotonia of both legs. Left arm weak, wasting of interossei in both hands.

In a few days he became semi-comatose, developed generalized tremors and became emaciated rapidly. Some of the deep reflexes that were absent returned. He showed spasmodic twitching and his breathing became shallow, his temperature rose higher and higher until it reached 107.2° F. at his death.

The temperature during the first three weeks was around 100° F., then it commenced to rise with fluctuation, but increasing steadily up to about 104° F., and during the last thirty-six hours climbed higher until it reached the maximum 107.2° F. at his death. Pulse and respiration kept pace with the temperature. During the last two days the pulse could not be counted, and the respirations were between 40 and 48.

Case 25. Fairly marked meningeal symptoms, radicular pains, pyramidal tract involvement, cranial nerve involvement, trophic changes, and mild psychic disturbances: I. R.; age twenty-three; single; American; Jewish; No. 17337. Admitted 9/26/21. Discharged 10/27/21. C. C.—Frontal headaches. Following strenuous day at the office, the patient developed a severe headache which up to then was mild all through the day. The headache was severe enough to keep him awake at night. It came suddenly and would not abate at any time.

9/26/21. Neurological examination—The patient looks very sick, complains of agonizing pain in frontal part of head, lower extremities, and back. Has had slight photophobia for the last twenty-four hours; he has also been more or less drowsy during the same period.

Pupils slightly irregular, react to light fairly well, but with suggestion of sluggishness; other pupillary reactions normal. Fundi negative. Ocular movements fairly normal, but there is suggestion of limitation of movement laterally. Tongue protrudes in mid-line. There is a suggestion of tremor. Biceps reflex fairly normal. Triceps both increased. Abdominal reflexes slightly overactive. Knee jerks and achilles markedly increased. Ankle clonus on both sides, more marked on left. No Babinski. Rigidity of neck fairly marked.

Kernig present on both sides. No tenderness of spine on percussion, no tenderness in any other part of body.

The patient claims that during the last thirty-six hours or so he has had dreamy states during which he felt himself riding and had some more or less clear presentations.

That very evening he developed a suggestive Babinski on the left, and difficult urination. Within the next two days the headache disappeared following lumbar puncture. The pupillary reactions were somewhat changed. Two days later he developed a tremor of the left hand. The ankle clonus was more marked on the left. The abdominal reflexes became diminished on the right. The patient was somewhat euphoric, insisted that he was not sick, and that he ought to be allowed out of bed. In a few days he developed radicular pains in various parts of the body. There appeared first mild paralysis of the left face, and in a few days it cleared up and the right face became paralyzed. Trophic changes of both feet were pronounced. Babinski was present on both sides, but more marked on left. Within eight days the facial palsy cleared up, the lower right abdominal reflex returned, and the ankle clonus on the right was much diminished, and the left pupil reacted somewhat sluggishly to light. On the day the patient left the hospital the following notation was made: The patient presents practically nothing new with the exception that he complains of radicular pain in the right chest. All the physical findings are the same. An occasional light vesicular rash is breaking out in various parts of the body.

The temperature was never over 100.5° F., and after the first few days, with one exception, when it rose to 99.5° F., it was around normal. Respiration and pulse practically normal throughout.

Reëxamination. 10/30/22. Just started to work one month ago. Claims that he feels fine at present. Suffered dizzy spells and severe headaches periodically about every month, but has not had them for the last three months. Had marked constipation.

Gets melancholic every once in a while, for three and four days at a time. Is irritable and the least trifle upsets him. "When under the depression feels like taking a gun and shooting his brains out." Used to have pains in the legs and feet, and finds that since he stopped eating meats he does not get the pains.

Shows slight mask face. Both pupils are markedly dilated. React well to all stimuli except sympathetic. Ocular movements normal. Tongue very tremulous, in mid-line. Definite weakness of left face—lower two branches. Right arm reflexes normal, left very much increased. Knee jerks markedly increased. Achilles very much increased. Ankle clonus on both sides, more marked on left. No tremor or rigidity of the hands.

Case 26. General infection, cranial nerve involvement, electric-like shocks, pyramidal tract involvement, delirium, and euphoria: Miss R. R.; age seventeen; American; Jewess; high school student. Admitted 3/17/22. Discharged 3/29/22. No. 6178. C. C.—Drowsiness, violent choreic movements, nervousness, and diplopia.

The patient had a cold two weeks ago. Last Monday night (five days ago) she had to leave school on account of seeing double, a headache, and feeling out of sorts. That night she could not sleep. She began to have choreic-like jerks of her entire body that came on suddenly, and caused her to rise up in bed and shriek with an indescribable sound. The next day she felt no better, slept but little that night and has not slept since. At present she sees double, and light hurts her eyes. She has had a high temperature once, but she does not know on what night.

3/16/22. Notes taken at patient's bedside before she was taken to hospital: About two weeks ago the patient had a cold. Since then she has had headache every day. For the last two nights she could not sleep. For the last few days, the brother noted that she was talking too much, and to-day she was not talking sensibly. She also had diplopia for the first time to-day. She complains of a severe jerking that wakes her up out of her sleep. The jerking is not confined to any part of the body. The patient is very voluble and somewhat confused.

Examination—Well nourished young girl. The eyelids are red, the pupils react promptly. There is definite nystagmus on looking laterally to either side. About every five minutes there occurs a typical electric-like choreic shock which practically doubles the body up, raises it out of bed, so that only the buttocks remain in contact with the bed, and the bed itself shakes from the rapid movement. This is accompanied by a shrill cry. Tongue is in mid-line. Slight weakness of right face. No rigidity of the neck. No tremor of hands. Arm reflexes not increased, and achilles jerks are moderately increased. No Babinski or ankle clonus. Abdominal reflexes increased on left. Slight hypertonia on left side of body. No Kernig. Temperature 100.2° F., pulse 84.

The patient is somewhat restless, tossing about. She is euphoric, and claims that she is not sick. She related that last night she imagined that she heard an iron ball rolling on the floor.

The next day she was admitted to the hospital, and when examined she was found definitely lethargic, but she would open her eyes for a few moments and again doze off. The nurse reported that during the night she was mildly delirious and kept the patients in the ward awake. In her delirium she talked mainly about her school work, her teacher, and her school mates.

Both pupils are irregular, but react well. There is paresis of the left external rectus. Nystagmus on looking to right. Tendency of tongue to deviate to left. Weakness of right face. Tremor of both hands, but more marked on right. Arm reflexes slightly increased on right, diminished on left. Abdominal reflexes diminished on right, present on left. Knee jerks diminished. Right achilles normal, left increased. No Babinski. No ankle clonus. Facial folds are becoming obliterated.

Two days later the choreic jerks stopped completely. The tongue

was deviated definitely to the left. There were trophic changes noticed in the feet. There was exhaustible ankle clonus and Babinski on right. The patient was very drowsy and irritable. On the next day she showed nystagmus in all directions, ptosis of both lips, and poor pupillary reaction to light on right. There was slight rigidity of neck. Right biceps reflex diminished. Right knee jerk more active than previously. No Babinski.

She stayed but a few days longer in the hospital, during which time she gradually grew worse, she was more lethargic, developed a definite ankle clonus and Babinski on right, and showed a masked face and marked tremor. The patient insisted, however, on being taken home, and her parents in spite of advice to the contrary removed her from the hospital. On the morning of the day she was removed from the hospital, she had a temperature of 104° F.

RETROSPECTIVE CASES

Case 27. Diplopia, headache, spasmodic tic which persists, and mild pyramidal tract involvement: J. Y.; age fifteen; fireman; American Italian. Admitted 6/1/20. Discharged 6/21/20. F. H.—Father and mother living and well. Three brothers and three sisters living and well. P. H.—None of usual diseases of childhood. No serious illness. Habits—Good. C. C.—Shortness of breath. Spasm of right side of face and neck.

During first week in April, 1920, he developed diplopia, headache, and digestive disturbances. His father states that he "was so weak in his bowels" that he had to go to bed for a week, and then the spasmodic twitchings came on, involving right neck, and lower part of face. Also had marked asthenia and shortness of breath.

6/3/20. Neurological examination—Tic of lower right facial muscles, and occasionally a deep inspiration. Pupils irregular, rigid, and unequal, right larger than left. React to light. Tic movement 50 per minute. Tongue in mid-line. Slight movement of right external rectus muscle, otherwise ocular movements good. Convergence poor. Fundi normal except that margins of left are somewhat blurred. Marked fine tremor of fingers. Abdominal reflexes active. Teeth crowded. Left incisor smaller than right. Knee jerks, right slightly increased, left normal. Babinski suggestive on both sides. Oppenheim suggestive on left. Ankle clonus negative.

Laboratory reports of no significance. Spinal fluid negative.

Case 28. Diplopia, delirium, marked psychic disturbances, reversed sleep and asthenia: M. E. M.; age fifteen; student; No. 734. Admitted 6/25/20. Discharged 7/13/20. F. H.—Mother and father living and well. Two brothers and one sister living and well. One brother dead—cause unknown. P. H.—Usual diseases of childhood. Diphtheria at nine. C. C.—Spells of sleeplessness, constant activity, and incessant talking.

About three months before admission, the patient developed insomnia, loss of appetite, diplopia for four days, and mild nocturnal

delirium. He would whistle all through the night, talk to himself or talk to pictures on the wall. He did many queer things, such as looking under the beds, locking the doors, and even picked his knuckles with a pen-knife until they bled.

This picture kept up for about two weeks, and then he attempted to go to school, but would fall asleep at his school work. He then developed peculiar mannerisms—such as blowing his nose all the time, or constantly repeating some funny statement that he heard for several days. He then developed marked asthenia.

Examination—Pupils about normal. Left lower face slightly flattened. Tongue in mid-line, but extremely restless. Knee jerks increased and unequal. Triceps jerks, right increased, left normal. Slight cyanosis of extremities. Moderate coarse tremor of extended hands, especially right. Thyroid moderately enlarged and firm. Pulse 60 in reclining position.

The patient has difficulty in falling asleep at night. Tosses about a great deal and falls asleep toward morning only and sleeps till noon. Pulse, respiration and temperature normal.

Case 29. Diplopia, meningeal symptoms, crossed paralysis, Milard-Gubler syndrome, fairly marked psychoneurotic symptoms, and residual organic signs are still present: A. G. H.; age twenty-one; American; laborer. Examined in March, 1922.

On September 20, 1918, while in the service, he took sick with what was diagnosed as meningitis. He had diplopia, left side hemiplegia, right facial paralysis, and was lethargic for six weeks.

His present chief complaints are: nervousness, restlessness, paralysis of the left arm, and a tendency to wander about.

Right pupil reacts well to all stimuli, the left pupil reacts sluggishly to light, but otherwise normal. Ocular movements normal. Tongue in mid-line. Paresis of the left face. Grip of left hand decidedly weak. Atrophy of muscles of left arm. All deep reflexes of the left arm are markedly increased. Deep reflexes of left leg increased. There is exhaustible ankle clonus on left. Right side normal. No sensory disturbance. The patient is extremely restless, irritable, and shows tendency to move about. He cannot sit still even during the examination.

Case 30. Slight pupillary involvement, nystagmus, and cerebellar symptoms: A. W. H.; age twenty-seven; American; machinist. Examined January, 1922. C. C.—“The nerves.” When he walks he feels as if he is going to fall forward. In July, 1920, he took sick with what was diagnosed epidemic encephalitis. On examination the pupils were found widely dilated. The left pupil had but a flicker of reaction to light, but all other reactions and that of the right pupil were normal. Ocular movements normal, except for a slight nystagmoid twitching on looking laterally, especially to right. Right fundus fairly normal except for slight blurring of nasal margins. Left fundus almost white and oblong atrophy. Slight weakness of left face.

Tongue very restless, but in mid-line. No palsy of palatal muscles. Coarse tremor of hands. Grip of both hands poor. Left jaw reflex slightly increased, right jaw reflex normal. Arm reflexes increased. Abdominal and cremasteric reflexes absent. Knee jerks increased and show pendulous swing, especially the left. Achilles increased. No Babinski or ankle clonus. No sensory disturbance. Slight swaying on taking the Romberg and mostly backward. Definite tremor of left foot on doing knee to heel test, but no ataxia. Pass pointing in left hand. Dysmetria is suggestive on left, but not definite. Adiadochokinesis of left slight.

Case 31. General infection, crossed paralysis, cerebellar symptoms and mild psychic disturbances: H. M. M.; age twenty-eight; American; gang leader P. R. R. Examined January, 1922. During October, 1920, he had "flu" and pneumonia. This was followed by Bell's palsy.

On examination the right pupil was found to be irregular. All pupillary reactions normal. Fairly marked nystagmus on looking to the right, otherwise ocular movements normal. Marked restlessness of the tongue and some fibrillation of the tongue muscles. Slight paresis of the left face. Jaw deviated to right. Arm reflexes increased, especially on the left. Knee jerks increased on the right, almost clonic response. No Babinski. Slight adiadochokinesis in the left hand. No pass pointing—finger to nose test. Some dysmetria present, especially in left hand. Some pass pointing on attempting to touch objects with left hand, but normal with the right hand. Gait good even with eyes closed.

Chin points to right, but the patient says that he has had this tendency ever since childhood, owing to some enlargement of the glands on the left side of the neck. Some hyperesthesia of the left face, otherwise no sensory disturbance. Hypotonia more marked in left hand. The patient is irritable and gets the "blues" frequently. X-ray of skull negative, Wassermann negative.

Case 32. General infection, myoclonic movements, cranial nerve involvement, Parkinsonian syndrome left, hemiplegia right, and bulbar myasthenia: Mrs. J. C.; age thirty; Slavish; married; No. 5945. Admitted 3/7/22. Discharged 4/4/22. C. C.—Tremor, difficulty in talking, constant headache, and drowsiness.

This patient was brought to the hospital two and a half years after the onset of her condition, which as yet was undiagnosed.

About two and a half years ago, the patient was delivered of her last baby. Shortly afterward she caught a cold which persisted for many weeks. About three months after delivery, or during the period shortly after recovery from her cold, she began to get drowsy, and she fell asleep for twenty-four hours without arousing. After she awoke, her arms and legs all began to move spontaneously in tremor-like movements, and she complained of double vision. For three weeks she had these clonic movements, continuously day and night,

also the diplopia. Several doctors had seen her, but nothing in the way of medication gave her relief. The legs quieted down after several weeks, but the arms have persisted in their movements ever since. About a year ago, she started to have difficulty in talking. She understands everything that is said to her, but cannot answer more than one or two words before there is a blocking. She has had headaches off and on since the onset of her movements. Sometimes it is greater than at other times, but it is a more or less constant symptom. At the onset of her illness, she had a temperature of 101 to 102, and has had a temperature sometimes since. She had never complained of any pain.

On examination she showed the following: She has a mask face, smiles unduly, eyes are bright. Talks barely above a whisper, and only says, "I no can talk." Pupils unequal. Right pupil does not react to light, left reacts very slightly. Consensual reaction same as direct light. Sympathetic and accommodation normal. There is nystagmoid twitch on looking to left. Fundi normal. Action of left inferior oblique muscle is slightly interfered with. Tongue red, edematous, deviated to right. Fairly marked facial weakness on right. Definite twitching of labial muscles, especially right. Left hand is held in the Parkinsonian position and shows a typical Parkinsonian tremor. Cog-wheel phenomena is present in both arms, more marked on left. Speech definitely bulbar in type. Arm reflexes increased equally. Abdominals are present, but not overactive. Babinski present and more marked on right. Exhaustible ankle clonus present, and more prolonged on left. Talking fatigues the patient, and after saying a few words, she can barely whisper.

Case 33. Diplopia, lethargy, paralysis, juvenile Parkinsonian syndrome, and psychic changes: R. J.; age thirteen; American-Italian.

This case was not diagnosed until brought to the neurological dispensary in March, 1922, with the following history: About one and a half years ago, the child got up one morning scared from a dream, complaining of seeing double. This lasted for about fifteen days, according to the patient's father. During this interval and for several weeks afterwards, the patient was dopy and sleepy—would go to sleep frequently and he had no energy or ambition to play. The father says that at no time did the child have any fever. He could not walk well after the sickness, and this has been getting worse lately.

On examination he presented the following: Typical Parkinsonian appearance. Slow speech. Definite cephaloptosis. The mouth partly open. There is definite shuffling gait, and the left foot especially comes down with a thud. There is an inclination to veer to the left when he walks. On starting to walk, he must run or he falls—propulsion.

On sitting down, he falls backward. Eyes normal except that pupillary reaction to light is somewhat sluggish. Tongue very

tremulous. Facial folds are obliterated. Definite weakness of right face. Grip of right hand is weak. Definite weakness of right arm. No atrophy of muscles in right arm. Cog-wheel phenomena definite in left arm, slight in right arm. Deep reflexes of left arm diminished. Biceps and triceps diminished on right, radio-periosteal present and active on right. Abdominal reflexes, very active, especially upper. Knee jerks on right diminished, on left present. Achilles present. Suggestive Babinski and definite fanning on right. Marked Babinski on left. Marked rigidity in right leg. Rigidity of the neck muscles present.

Mentally the boy changed considerably. Since he took sick he cannot learn, although he was exceptionally bright before. Smiles in a silly manner all the time. He has no insight at all.

Case 34. Mild infection, diplopia, pressure sensation, and Parkinsonian syndrome one and a half years after onset of the condition, following an operation: Mrs. F. H.; age thirty-six; Austrian Jewess; housework. Examination in May, 1921. C. C.—Weakness in left arm and tremor. Under the psychic influence of facing the doctor, the arm shakes a great deal more. Sensation of hot flushes. This condition existed only for the last three months. P. H.—Two years ago she had a pressure sensation in the head. Glasses by E. improved her condition. She saw double for one day. Was in bed at the time for five or six days. Felt sleepy. Had headache at the time in the temporal region. It took about two or three weeks before she really came to herself. Three months ago, she had an operation on her uterus and on leg, for varicose veins. Since the operation the arm troubled her.

Neurological examination—There is a mask face. Pupils irregular, contracted, and react to light very sluggishly. Contract better to accommodation. Ocular movements normal. Tongue in mid-line and tremulous. Slight weakness of right face. Thyroid enlarged, particularly isthmus. Marked Parkinsonian tremor in left hand. Cog-wheel rigidity not very marked in left arm. Arm reflexes increased, left more than right. Knee jerks increased more on left, and left leg feels fairly rigid. Both achilles increased. No Babinski or ankle clonus. Sensory response not reliable.

Case 35. Ambulatory case, lethargy, mild cranial nerve and pyramidal trace involvement: J. W.; age twenty-five; single; grocer; Russian Jew. C. C.—Lethargy for the last two weeks. Had diplopia for about a day or so. Headache for a few days. No dizziness. Worked all week but felt sleepy. The day before, he came home at three and fell asleep for three hours. Had difficulty with his bowels last week. Was constipated.

Neurological examination—Pupils: right is triangular, reacts sluggishly; left slightly irregular and reacts fairly well. Slight paresis of left external rectus. Nystagmoid twitch on looking to right. Very slight deviation of tongue to right. Apparent weakness of right face.

Knee jerks and achilles very much increased. Some slight rigidity of muscles.

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SOCIETY PROCEEDINGS

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

REGULAR MEETING DECEMBER 6, 1923, AT THE BOSTON MEDICAL
LIBRARY

F. K. HALLOCK, M.D., President, in the Chair

HEAD NYSTAGMUS IN MAN, WITH A REPORT OF A CASE

C. L. WOOLSEY, M.D.

Head nystagmus in man has frequently been observed by otologists in individuals who have been afflicted with a fistula into the labyrinth. On the other hand, comparatively few cases have been reported of head nystagmus in man not due to labyrinthine fistula. Mygrind in a recent article on "Head Nystagmus in Man" calls attention to the classical experiments on pigeons causing rhythmic movements of the head. He is convinced that head nystagmus in the human is a normal vestibular reflex, but he concludes that "head nystagmus in man is of the greatest rarity." I do not agree with that statement. I believe that head nystagmus in man occurs frequently in lesions encroaching upon the vestibular system. A number of cases of head nystagmus, not due to fistula into the labyrinth, have been observed in the Nerve Clinic of the Massachusetts General Hospital. Recently I have seen a case that is unquestionably one of head nystagmus due to a pontine lesion encroaching on the posterior longitudinal bundle and the vestibular nuclei. I will give you a brief summary of the case.

Oct. 1923. Nerve Dept. OPD, Mass. Gen. Hospital. G. S., age twelve. He was about the average in intelligence, quite diligent, and good natured. In May, 1923, he became listless, lazy and had decided memory defect. He was lethargic and slept morning and night. Vision began to fail, and diplopia was distressing. He experienced vertigo (both objective and subjective), accompanied by nausea and vomiting. Later, during the course of the disease, insomnia superseded the lethargic state. He walked toward the right anterior quadrant. Eye nystagmus, at times horizontal but usually vertical, was noted by the mother. She also noted that soon after the onset of the eye, a head movement "just like the eye movement" appeared.

This patient was sent by the local doctor to the Boston Psychopathic Hospital, and thence to the Worcester State Hospital, which in turn, sent him to Dr. Harvey Cushing's clinic, and later to the

Massachusetts General Hospital, where I saw him. The patient has definite head nystagmus that is usually vertical in type, with the slow component downward and forward and the quick phase upward and backward. His head nystagmus may be rotary, diagonal, and at times horizontal. It is interesting to note that the head nystagmus is synchronous with the eye nystagmus and always like it.

The caloric tests are also interesting, and indicate a pontine lesion of the multiple sclerotic type. In July, 1923, at the Boston Psychopathic Hospital his spinal fluid Wassermann was negative, but the gold sol showed a parietic curve. A short time later, the fluid was examined at the Peter Bent Brigham Hospital and, although the gold sol curve was of the parietic type, it was not so intensely so as when taken at the Psychopathic Hospital. Analysis of the spinal fluid at the Massachusetts General Hospital on two subsequent occasions was entirely negative, the three tests indicating a fluid, first abnormal, that later became normal.

The whole picture is one of lethargic encephalitis complicated by a lesion involving pontine and vestibulospinal tracts of the vestibular system, with a resultant head and eye nystagmus that are alike and synchronous.

A complete report of the case will appear at a subsequent time.

Discussion: Dr. E. W. Taylor: This is obviously an extremely difficult communication to discuss because I think most of us are not sufficiently familiar with the complicated problems which the labyrinth presents. Dr. Woolsey's work, which it has been my privilege to follow in a somewhat superficial way, has been exceedingly clever and painstaking, and I feel that he has added something definite to our knowledge of this very intricate subject, and particularly to head nystagmus or head movements which he is right in saying we have been inclined to put in the category of tics, or habit spasms, or possibly explaining them on some psychogenic basis. This opinion he has done much to disprove.

Dr. F. K. Hallock: I might inquire if this boy had ever witnessed the performances of the pigeons, with the question whether there is any imitative feature in the case.

Dr. Taylor: The diagnosis in this case was very interesting. At the Psychopathic Hospital the boy was diagnosed as juvenile general paresis, and apparently it had much to bear this out, the gold sol curve, for example. At Worcester they were very much puzzled; and Dr. Cushing, unless I am mistaken, made a diagnosis of encephalitis with a pontine lesion.

Dr. H. C. Solomon: Relative to the diagnosis made at the Psychopathic Hospital: I was in California at the time. I saw the patient later at the Massachusetts General Hospital and noticed that it was stated the Psychopathic Hospital physicians had made a diagnosis of juvenile general paresis. I asked several of the men what the diagnosis was, and they all told me it was encapalitis. It was one of those clerical errors where the diagnosis had been neglected and not put on the record.

Dr. H. Cushing: I may make almost the same apology in regard

to my connection with the case because I had no recollection of having made the correct diagnosis, but I am glad to stand just for a moment to add my word of congratulation to Dr. Woolsey and appreciation of the work he has been doing all these years. He has been wrapt up in it and has already done a great deal, and is bound to carry it far, I am sure. I hardly feel that in cases of this sort surgeons are called upon to do much more than decide whether it is or is not a surgical case. About all we could say regarding this boy was "These are cerebellar symptoms and may be due to a cerebellar lesion"; but there was no evidence of tumor, and I think the diagnosis of juvenile paresis had come over from the Psychopathic Hospital. I don't know which of us suggested encephalitis. I only fear that we are going to make that diagnosis too often and that a diagnosis of an encephalitic syndrome may become a blanket for a great many conditions of another nature.

Dr. Woolsey: In answer to Dr. Hallock, I cannot say as to the boy's seeing other pigeons; I know he did not see any of mine.

In regard to the work referred to by Dr. Cushing, the tests are severe and my object was and is to classify these syndromes so that they will be easily understood. We have some syndromes relating to quadrants of the body, and patients complain that they have a tendency to fall or are drawn into one or more of these quadrants, I am attempting to prove that those movements are due to vestibular lesions; that we do have head nystagmus in man; that it is relatively common; that we have had 30 to 40 cases of head nystagmus synchronous with eye nystagmus. Torticollis is frequently due to some lesion in the vestibular apparatus (a lesion at some definite point), and needless operations on the neck and needless treatment would be avoided if we did not say that certain movements of the head were tics and of psychogenic origin. Perhaps they are not, and in the years to come somebody will classify these syndromes so that it will not be necessary to do the Barany tests on every patient we meet. It is with this in view that I am conducting my experiments.

RADIOGRAPHY FOLLOWING THE INJECTION OF LIPIODOL INTO THE SPINAL SUBARACHNOID SPACE

JAMES B. AYER and WILLIAM J. MIXTER

One of us (J. B. A.) has previously presented before this Society a method for the determination of spinal subarachnoid block by means of dynamic studies in connection with lumbar puncture, and more specially combined lumbar and cistern puncture. While of great aid in separating spinal cord lesions due to compression from degenerative diseases of the cord, this method does not give evidence of the location of the block. The object of the employment of lipiodol is not only to demonstrate a block but also to localize it.

Some time ago Dr. H. T. Patrick wrote us of the method which he had seen used by Sicard with success, upon which two articles have been published, one by Sicard and Forestier (*Presse Medicale*,

1923, No. 31, p. 493) the other by Percy Sargent in an August number of the *British Medical Journal*. The former paper deals with the use of lipiodol in a number of different locations, but emphasizes its use in (1) the subarachnoid space, and (2) the epidural space. The object of both of these types of injection is to locate an intraspinal tumor or level of compression of the cord from other cause. The substance used—lipiodol—is a poppy-seed oil containing chemically combined iodine to the amount of 0.54 grams per cubic centimeter. It is said to be nonirritant in the subarachnoid space, and is very opaque to x-rays. Sicard recommends 2 c.c. for subarachnoid injection, and 4 c.c. for epidural use. While the article by Sicard and Forestier is illustrated by numerous diagrams representing the oil drops arrested in the neighborhood of obstructions, the short paper by Sargent is accompanied by x-ray reproductions of three cases in which tumors have been localized and by means of the oil, and successfully removed. In Sargent's cases the oil was introduced by cisternal puncture, and allowed to settle to the level of the spinal obstruction.

We have for three months experimented with lipiodol and with a number of similar oils made up for us by Dr. J. L. Stoddard, Chemist at the Massachusetts General Hospital. This experimental work was performed on cats, the oil being injected in 1 c.c. and 1.5 c.c. amounts into the cisterna magna, replacing an equal amount of fluid withdrawn. More recently we have employed lipiodol in two patients, hopeless paraplegics from metastatic spinal disease. These injections were into the lumbar sac, 2 c.c. and 4 c.c. being used respectively.

Our experience with the French preparation of lipiodol may be briefly summarized as follows:

(1) The oil is impervious to x-rays, even a small droplet casting a shadow which is not to be confused with normal or pathological tissues.

(2) The oil is disseminated slowly throughout the spinal and cerebral subarachnoid spaces of the cat under normal conditions.

(3) Absorption is extremely slow. One animal injected nine weeks ago still shows most of the oil in the subarachnoid space, much as seen after its introduction.

(4) The irritability of the oil was obvious in all of the six animals injected with the French oil. The cats were usually "groggy" for one, two or three days. As an index of irritation of the meninges it may be noted that in one cat the cell count in the spinal fluid was 4,420, mostly polymorphonuclear leucocytes, on the third day after oil injection. A cat injected yesterday shows a fluid to-day containing 2,700 cells. One animal went into convulsions immediately after an injection and died; at necropsy there was no evidence that the needle had injured the brain.

The irritability of the oil in the two patients was not controlled by reexamination of the spinal fluid, nor are neurological symptoms significant, because of the completeness of the paralysis in each case. One, however, ran a mildly elevated temperature, otherwise unexplained, for nearly a week after the injection.

(5) While we cannot be certain that in our patients the block was localized by the lipiodol, as operation was not performed in either case, it is probable that the oil correctly indicated the site of block, in that it was visible nearly up to the level as clinically determined, and did not extend above it.

From our present slight experience we feel that while lipiodol is an agent of probable value in the localization of spinal block, its very considerable irritability within the spinal subarachnoid space must be reckoned with; also that its very slow absorption may be an important disadvantage. It would seem, therefore, unwise to use this diagnostic procedure as a routine measure, but to reserve it for cases in which clinical and other laboratory methods are insufficient.

Dr. Mixer: Dr. Ayer has stated everything that I should want to say except this: I should rather make it a little stronger that I am distinctly dissatisfied with this procedure as it stands. I am satisfied that it has a definite application if we can get a nonabsorbable fluid that is opaque, which will rise fairly rapidly instead of disseminating.

Discussion: Dr. P. Bailey: I am familiar with the French experimenters. I was present when Sicard read the article referred to. We have some lipiodol locked up at the Brigham Hospital which we have not used.

THE ANATOMY OF THE INTRACRANIAL SUBARACHNOID SPACES

DR. HOWARD C. NAFFZIGER of San Francisco, by invitation

In this paper Dr. Naffziger discussed the subarachnoid spaces on the basis of casts and diagrams with lantern demonstration. The research is to be published later.

Dr. H. C. Solomon: From the lantern slides it was not clear to me what happens to the ends of these spaces, as they seem to end blindly; that there was not a complete circulation; this obviously is not Dr. Naffziger's idea. What relation have these casts with the sinuses? One would assume that these injections would lead somewhere into an entrance into the sinuses. If the velum that separates the internal cisterna from the lateral ventricles is in life so thin and is simply a parchment, as it were, separating two bodies of fluid, with the tremendous changes of pressure that must occur in certain cases of hydrocephalus and other cases of shock, fall, and blows, why do we not get injury to this and to the brain? He mentions in one instance the possibility of this having occurred and circulation reestablished in hydrocephalus. Isn't that likely to happen in brain trauma? Why doesn't it happen more frequently? Is this very thin membrane acting as a dialyzing filter to allow exchange of fluid?

Dr. W. J. Mixer: This has been a very interesting subject to me. It was brought to my attention last summer at the meeting of the American Medical Association in San Francisco, and at that time I saw Dr. Naffziger's casts. I was tremendously struck by the extent of these subarachnoid spaces as shown by the casts. I think we must bear in mind in our discussion, however, that they are in life to a certain extent potential spaces rather than real spaces, and

that very probably some of these thin membranes are actually thin veils between the ventricle and the subarachnoid space and probably pushed up against the underlying brain tissue so that in life we would find no space at all. The subject of hydrocephalus is one of much interest. The position of the block in the cerebrospinal fluid circulation is so variable that any additional information which we can have as to that circulation, either physiological or anatomical, is of great value. Such a study as this might give us a great deal of information in dealing with a hydrocephalus caused by the blocking of the iter. Whether that information can be utilized or not we do not know as yet. Dr. Naffziger has given us a hint of his future plan on that. I feel that the position of the blocking mechanism is so variable that we must look for different operative measures for our different types of hydrocephalus, and he must carry his work on. He has developed something that to my mind is very valuable, and I hope to see him carry it on and give us a definite treatment for hydrocephalus.

Dr. J. W. Courtney: There is another point of interest; that is the possibility this demonstration affords by demonstrating the various routes taken in hydrocephalus that we may have the explanation of the various forms of encephalitis lethargica.

Dr. Addison, Rochester, Minn.: There is very little to add to what has already been said, except to congratulate Dr. Naffziger on the work. All of us doing neurological work are interested in hydrocephalus. All methods so far have proved unsatisfactory, and if Dr. Naffziger has devised something to help us in hydrocephalus, we shall appreciate it. The study has meant much earnest and hard work and discouragement, I am sure, and I heartily congratulate him on his studies.

Dr. Ernest Sachs of St. Louis: I am one of the fortunate people who have seen the original models, which, as Dr. Naffziger says, give a much clearer conception of these spaces. Since he has pointed this out, I have made one observation in a case; in opening the ventricle I saw very clearly an extremely thin membrane which was evidently the point where this internal subarachnoid space lay close to the ventricle. Whether that occurs in all cases of internal hydrocephalus, I am unable to say. In that particular instance, it would be a perfectly simple thing to make a hole through that membrane. It would be interesting to know in what portion of the ventricle this thinning occurs. I suppose Dr. Naffziger will work that out later. This particular point was right in the center of the optic thalamus. Whether the thinning always occurs there or not, I do not know, and perhaps in his concluding remarks he may throw some light on that.

Dr. Naffziger: Dr. Solomon asked a number of questions which I cannot answer. The velum referred to is a very thin membrane and is very closely supported by the basal nuclei and approximately in the situation noted by Dr. Sachs. In answer to Dr. Mixter, I think we can say these are actual spaces.

NEW YORK NEUROLOGICAL SOCIETY

THE FOUR HUNDRED AND EIGHTH REGULAR MEETING OF THE
SOCIETY WAS HELD TUESDAY, DECEMBER 4, 1923,

DR. E. G. ZABRISKIE PRESIDING

MYOTONIA

Dr. M. Neustaedter: This boy, now eleven years, was presented, five years ago, with a diagnosis of Oppenheim's disease. The parents are cousins. Though the case is one of myotonia, there is no response to either galvanic or faradic current in the lower extremities. There is a marked hypotonia, but no muscle wasting. The Wassermann, in blood and spinal fluid, is negative. The involvement is simply one of the lower extremities. The boy improved remarkably until he had an attack of double pneumonia. The loss of response to galvanism and to faradism is hard to account for. He has no sensory disturbances. I had also two other cases under observation. These patients died. In these all muscles were involved, but there was no wasting of the muscles.

JUVENILE PARESIS

Charles Rosenheck said some time ago it was his privilege to present before this Society a clinical study of juvenile tabes. Its etiology and extreme rarity as compared with its incidence in later life, the clinical phenomena, the differences in behavior between the juvenile and adult types of the affection, were dwelt upon and a number of deductions and conclusions formulated.

Case I. Female, age seventeen, and elder of two, the other having died at the age of five from an acute surgical condition. Two weeks after the birth of the patient a rash developed which yielded to mercurial rubbings. She walked rather late (two years), teethed early and talked at the normal period. Besides the minor infantile affections, she had no serious ailment. She began school at seven but was "left back" a number of times. At the age of sixteen the teacher, struck by her apathy and lack of understanding, referred her to a clinic where a diagnosis of neuro-lues was made.

According to the mother, the patient has for a considerable time shown marked hebetude, but there has at no time been any gross disturbance in conduct, judgment, orientation or memory. In fact her mother thinks her memory is quite good. She is docile, accepts all orders without protest and is well-behaved. A year ago she developed lancinating pains in the lower extremities and occasional headaches. Quite recently an apoplectiform seizure produced an aphasia and confusional state which disappeared in a few days.

Examination showed pupillary rigidity, negative fundi, tremor of

hands, tongue, lips and facial muscles, slight Romberg, hyperactive deep and superficial reflexes.

The blood Wassermann is strongly positive and the spinal serology is typically parietic.

Case II. Patient is a boy, nine years of age, and is the second child, first having died of an intercurrent affection. Three other children, of eight, seven, and four years, are apparently in good health and show negative blood Wassermanns. There is no history of miscarriages. Birth and early development were apparently normal. At five months, however, he had a rash on his legs which disappeared after medication. He walked and talked and showed normal mental development at proper time. He began school at six and seemed to get along well for about one year, then his teacher noticed his inability to grasp rudimentary class work. His memory also showed grave defects inasmuch as he would forget to come home and frequently was found wandering away in an aimless sort of manner. This state of affairs has continued for past two years. About one and a half years ago, speech defect became evident and increasing failure of memory and deterioration manifested themselves. He wets and soils himself quite often, expresses no wishes or desires and shows apparently no spontaneity in thought or action.

The examination revealed a gait which was slightly ataxic, a slurring parietic speech, pupillary rigidity, labio-lingual tremors and hyperactive, deep and superficial reflexes. The blood Wassermann was positive and the spinal serology was typically parietic. The father shows a positive Wassermann. The mother refused to have a blood test performed.

Comment: Juvenile general paresis occurs more frequently than we have been aware of.

That quite a number of cases are apparently the formes frustes. Inasmuch as these cases are usually placed in the indeterminate neuroleptic group, this may in a measure account for their lack of recognition.

Juvenile general paralysis is the product of a prolific and degenerate stock plus a congenitally acquired syphilis.

The clinical picture of general paralysis in childhood or youth is rarely as typical as that of the adult. This is obviously due to the incomplete cerebral development and insufficient mental acquisitions. In the main, however, the symptomatology differs very little from the adult form. Two marked-features are, however, present in the juvenile form which are worthy of mention. These are entire absence of grandiose ideas and early and progressive deterioration.

Discussion: Dr. Jos. Smith said: I think that a thorough mental examination is necessary before making a diagnosis. Progressive deterioration is not enough, or bad memory. Is the memory really affected so that they do not remember dates, or is it a general lack of attention. I do not favor a diagnosis of paresis.

Dr. J. H. Globus said: I would like to ask why the doctor made a diagnosis of general paresis. What is there in these cases more

than congenital lues with involvement of the central nervous system? What justifies a diagnosis of juvenile paresis?

Dr. L. H. Cornwall said: There does not seem to be a great amount of deterioration in the girl, considering the length of time she has had this condition. How long has Dr. Rosenheck had these cases under observation? What was the treatment and what was the effect of treatment? The young boy does not show great deterioration and I think we might doubt the fact of general paresis. One might even expect considerable improvement in the girl's case. I should like to ask about the serology of the parents. The fact that juvenile paresis does not show the same mental changes as in adults is because the two have not the same psychic background. I think these are straight cases of congenital lues and the young boy has probably parenchymatous syphilis in the cells of the cerebral cortex. Whether these cases are paretic is extremely open to doubt.

Dr. E. D. Friedman said: I have seen tabo-paresis in a boy of fourteen, corresponding to the type of the younger patient. It started with a tabetic picture with loss of reflexes, then rapid mental deterioration, convulsive seizures and optic atrophy. The spinal fluid was positive. That case had the features that Dr. Rosenheck has shown here: involvement of the base of the brain and hypothalamic region, which is an important vegetative center. The first patient, the young girl, is not typical dementia paralytica. The convulsive seizure suggests paresis. The boy is said to show no grandiose notions, but the psychic content of the child's mind is not capable of building up an elaborate structure, as in adults. The length of the disease is easily explained as it takes ten or twelve years for congenital lues to declare itself in the nervous system. In this boy there does not seem to be evidences of Hutchinson's teeth, or of keratitis.

Dr. P. R. Lehrman said: Euphoria in paresis is rare rather than common. Psychiatrists have difficulty in pointing it out in this syndrome. I doubt very much if the girl had paresis. A thorough mental test should be made.

Dr. I. J. Sands said: In regard to grandiose notions, these children use very bad cuss words, which in a way is the childish substitute, and you can tell by the bad language they use the way they are affected. The instinctive side of their nature is very precocious. Intelligence tests on these patients are very misleading, because they are far ahead in certain things. Judgment is as a rule weak. Treatment does not do any good in these cases.

Dr. Rosenheck (closing) said: I am grateful for the discussion and sorry I could not show you what dementia paralytica should show. Adult cases do not always show clear cut pictures, which should be emphasized. Juvenile paresis is a long drawn out affair, with slow deterioration. I would emphasize what Dr. Sands said about intelligence tests. The serology is negative in the girl's parents, but she had a luetic rash at birth. The boy's father shows a 3 plus Wassermann. I have no doubt that on the East Side, where Dr. Sands works, the children use pretty bad language.

INVOLUNTARY MOVEMENTS: THEIR UNUSUAL ASSOCIATION AND RELATION OF DYSKINETIC AND EXTRAPYRAMIDAL SYNDROME TO THE PHENOMENA OF DECEREBRATE RIGIDITY

Drs. S. Brock and I. S. Wechsler (author's abstract): The following cases were shown from the Neurological Service of Montefiore Hospital: *Case I.* A girl of eighteen, who developed a choreiform, Parkinsonian, and tic-like movement, after epidemic encephalitis. There was also an hysterical astasia abasia present; and one year after the acute encephalitis she showed bilateral ptosis, nystagmus and slight weakness of the right face in addition to the hyperkinetic phenomena above mentioned.

Case II. Married woman, twenty-eight, who suddenly developed an involuntary movement confined to the musculature of the right foot, which consists of a slow, dystonia-like fanning of the toes by plantar flexion. This interferes with walking. Occasionally the toes exhibit a tremulous movement. There is no history of antecedent disease. No other symptoms or signs are present, and the condition has remained stationary for the past three years. The patient is not of the functional type.

Case III. Boy, eleven, Italian, revealing a remarkable dyskinetic syndrome, following epidemic encephalitis, belonging to the dystonia group, sequential in nature and limited to the head and neck musculature. There are recurring waves of movement in which the head is greatly retracted, the back becomes opisthotonic, the right hand becomes hyperpronated, and the right foot assumes an equinus position. The whole picture forms an exquisite example of decerebrate rigidity. During the seizures (which vary considerably in frequency and intensity) there is loss of associated swing in the upper extremities. The pupils are unequal, irregular and react poorly to light and accommodation. There is a fine, rapid, lateral nystagmus and a marked vertical nystagmus. Conveyance is poorly done. The tongue deviates to the left and shows coarse tremors and fine fibrillations. There is hypertrophy of the sternomastoid and shoulder girdle musculature. During an attack these muscles are hypertonic. A respiratory grunt occurs during the seizure. There is bilateral Babinski present, with equal, lively knee and ankle jerks. There is no ankle clonus; the abdominal and cremasteric reflexes are equal and active. There are no cerebellar or sensory signs. The variety of signs is indicative of the diffuse multiplicity of lesions from basal ganglia to medulla.

These cases indicate the peculiar associations and fragmentations of involuntary movements and reflect the serious inadequacy of the present nomenclature.

Objection is taken to Walshe's stand (expressed in his article in the July, 1923, number of *Arch. of Neurol. and Psych.*) in which he limits the phenomena of decerebrate rigidity to *lesions of the pyramidal pathways*, excluding the extrapyramidal syndromes. The

greatest objection to this concept is that a hemiplegic lower extremity, due to a lesion of the pyramidal tract in the cervical or upper lumbar spinal cord is the same in nature, as regards posture, reflexes, as the plegic lower extremity produced by a mid-brain interruption of the pyramidal tract. Of what avail is that mechanism in the ponto-mesencephalic region, the release of which gives us experimental extensor rigidity? Secondly, Warner and Olmstead (*Brain*, Vol. 46, part II, July, 1923) have shown the significance of the ponto-fronto cerebellar pathway, as the inhibitor of decerebrate posture in cats. This tract rather than the pyramidal tract deserves attention in this connection. Replying to Walshe's criticism that no one has observed the presence of involuntary movements in the decerebrate animal, we would point out the unfairness of comparison between an *animal* in which a carefully planned surgical experiment has produced a *sole* effect, *i.e.*, decerebrate rigidity, and *man*, in whom inflammatory and heredo-degenerative diseases of long development and standing have manifested decerebrate rigidity phenomena which are only partially developed, are secondary, and do not occupy the foreground of the clinical picture. Lastly, Meyers (*Archiv. Neurol. and Psychiat.*, 8:4, 383, October, 1922) has made a careful clinical and pathological report of two cases, which showed Magnus-DeKleijn and decerebrate rigidity phenomena. In one, clinical and pathological investigation disclosed *no lesion of the pyramidal pathway*.

We feel that one does observe phenomena showing the pattern of decerebrate rigidity in dyskinetic and extrapyramidal syndromes, and that no one has conclusively shown that the *quality* of this extensor postural tonus must be the *specific* hypertonus of pyramidal tract lesions.

Discussion: Dr. Henry A. Riley said: The iconoclastic spirit seems to be rife this evening. I protest against including the woman with the foot movement, in this class of cases. Her disturbance seems to me to be of functional type. Watching the movements, either all the toes come up or go down, and I cannot conceive where such a movement can originate, except the cortex. No disturbance in other sites could cause motor phenomena of that type. The other case is extremely interesting.

Dr. Wechsler said: There is not much iconoclastic spirit in this paper. It is in answer to Walshe's criticism on decerebrate rigidity. This term is used too loosely, but it may be applied clinically to the cases presented. Dr. Riley's criticism is only partly justifiable, hysteria lasting three years would be unusual. We believe there is invasion of the basal ganglion. I believe it is dystonia, but perhaps mental examination and analysis might reveal other factors. If so, the patient would be excluded from this group.

CURRENT LITERATURE

I. VEGETATIVE NEUROLOGY.

2. ENDOCRINOPATHIES

Houssay, Galán, and Negrete. PITUITARY EXTRACT AND DIURESIS.
[Revista de la Asociación Méd. Argentina, January-March, 1921.]

This paper records experiments made in dogs and rabbits by subcutaneous and intravenous injections of a saline extract, acidified with acetic acid (0.25 per cent) of the posterior lobe of the pituitary of the ox. In the rabbit a fleeting oliguria occurred, but if the animals received abundant amounts of water, administration of the extract had no effect on the quantity of urine passed in the twenty-four hours. In the dog a diuretic effect was noticed which lasted some hours, but had no influence on the mean secretion measured during twenty-four hours; the diuresis consequent on increased ingestion of water appeared to be diminished by the giving of the pituitary extract. Houssay, Carulla, and Romaña (Ibid.) found that in twelve out of thirty dogs polyuria followed experimental puncture of the infundibulo-peduncular region of the brain; the same result followed in four dogs in which the hypophysis had been removed.

Jansen and Broekman. HEREDITARY DIABETES INSIPIDUS. [Nederl. Tijdschr. v. Geneesk., May 7, 1921. B. M. J.]

These authors report fourteen cases of diabetes insipidus which occurred in five generations except in the second, in which apparently no member was affected. Four of the cases were males and ten females, contrary to the general rule that hereditary diabetes insipidus is commonest in the male sex. They allude to the pedigree compiled by Weil, senior and junior, of five generations consisting of 220 persons, of whom 35 (21 men and 14 women) had diabetes insipidus. They have also collected eleven other cases of hereditary diabetes insipidus reported by Oris, Pain, Gee, Marinesco, Lauritzen, McIlraith, Jasse, Clay, Lacombe, Hewson, and Lancereaux respectively. As in the cases reported by Weil senior, who describes hereditary diabetes insipidus as a "healthy disease" (*gesunde krankheit*), the writers' patients were able to follow their occupation, felt quite well, and reached old age. The symptoms usually developed in infancy, sometimes later, became more marked about the twenty-fifth year, and then diminished. The abundant excretion of water had no effect upon the heart. The blood pressure was normal. Weil senior maintained that the hereditary form of diabetes insipidus should be separated from the acquired form, but

the present writers hold that there are no distinguishing features except heredity. Weil suggests that there is a difference in prognosis, but, as the writers point out, in many cases of diabetes insipidus in which cerebral tumor, cerebral syphilis, tuberculous meningitis, etc., can be excluded, the prognosis is also favorable. There are thus many resemblances between diabetes insipidus and diabetes mellitus. In both there is an hereditary and a nonhereditary form, in both there is an endocrine factor which may come into play (hypophysis, pancreas, etc.), and in both it is possible to produce the disease by experiments on animals. It is further noteworthy that in Graves's disease both polyuria and glycosuria may occur; that in the same family there may be cases of both diabetes insipidus and diabetes mellitus; and, lastly, that recovery from diabetes mellitus may be followed by diabetes insipidus.

Monakow, P. v. PATHOLOGY OF THE HYPOPHYSIS. [Schw. Arch. f. Neur. u. Psych., Vol. VIII.]

This is a report of a case of clinically hypophysial obesity with abnormal distribution of fat, reduction of metabolism, increased carbohydrate tolerance, abnormally slight reaction to adrenalin and thyroid extract. There was marked atrophy of the generative glands and of the Leydig cells, decline of secondary sex characteristics, oliguria and mental apathy. Pathological substances appeared in the urine only ten days before death. Histopathologically there were degenerative changes in the tortuous uriniferous tubules with almost complete atrophy of the anterior lobe and of the pars intermedia of the hypophysis while the posterior lobe was unaffected.

Samaja. PITUITARY ANOMALIES IN TWINS. [Chir. d. Org. d. Mor., December 1921, V, No. 6. J. A. M. A.]

The sella turcica is abnormally small in both the young men, but one has developed to above the normal height, while a congenital deformity of the legs in his twin shortens his height materially. Both at sixteen are otherwise well developed. Samaja urges study of the sella turcica in twins.

Verger, H., Massias, C., and Auriat, G. INCREASED TOLERANCE OF CARBOHYDRATES AND ABSENCE OF THE REACTION TO PITUITARY POSTERIOR LOBE EXTRACT IN AN ACROMEGALIC. [Compt. Rend. Soc. de Biol., LXXXVII, June 17, 1922, 197.]

A woman, thirty-six, had had for thirteen years epileptic attacks and acromegaly limited to the face, with hirsutism. Radiography of the skull showed an enlarged sella. There had never been adiposity, gigantism, genital troubles, polyuria nor glycosuria. Glucose was given for six days, in doses increasing daily by 50 grm., beginning with 100 grm. and ending with 300; no glycosuria followed. Even after intramuscular injection of 0.20 grm. of extract of the posterior pituitary

lobe, and after a test meal of carbohydrates, no changes were seen in pulse or arterial tension, and the quantity of urine was not influenced. Here, then, there was dysfunction of both lobes of the pituitary, that of the anterior being shown by the acromegaly, and that of the posterior by the exaggerated carbohydrate tolerance and absence of the reaction to the injection of pituitary posterior lobe extract. [Leonard J. Kidd.]

Davis, J. S. DIABETES INSIPIDUS. [Virginia Medical Monthly, May 1921.]

This article is chiefly a report of six cases of the disease seen in the last nine years, in five of which there was more or less evidence of intracranial pathology, the last one especially incriminating the pituitary gland. All but one (a suicide) are still alive and two apparently relieved. All are white and all males except one who had dystrophia adiposo genitalis which yielded more to pituitary therapy than the polyuria did in her case. The most striking instance seemed to follow lethargic encephalitis and showed symptoms of dyspituitarism with an abnormal sella turcica. This case was very fully reported and very fully investigated and described. Temporary relief was afforded by pituitary extract, which, however, so blurred vision and added to the pitiable nervousness that the patient refused further treatment along that line. All physical examinations were negative as were also the blood and spinal fluid Wassermanns. Lumbar puncture gave only transient relief, reducing urinary output from 17,000 c.c. to 7,000 c.c. for one day, possibly because the headache entailed kept him from drinking his usual amount of water. He generally excreted from 50 to 500 c.c. more water than he drank. The urinary specific gravity was never raised but once above 1004. The temporary relief from pituitary therapy have been due to the diminished absorption of water which has been claimed as its chief result, but I was unable to follow it further on account of the patient's obstinate refusal. Bromides and valerianate of zinc gave better results than any other means employed and in one instance apparently afforded a complete cure. The headaches, eye symptoms, disturbed sexuality, and X-ray findings in the last case suggest dyspituitarism as being in some measure responsible for the trouble. Relapses often occur and it is doubtful except in syphilitic forms if final restoration is ever obtained. Elsner regards it as absolutely incurable, though certainly spontaneous recoveries occur and amelioration with a tolerable existence is not uncommon. Even in the most favorable cases medication and a hygienic life will have to be kept up carefully and indefinitely. [Author's abstract.]

Shapiro and Marine. SUPRARENAL GLAND IN EXOPHTHALMIC GOITER. [Endocrinology, November 1921, V, No. 6. J. A. M. A.]

The case of exophthalmic goiter reported by Shapiro and Marine presented several unusual features in addition to the classical manifesta-

tions of profound asthenia, emaciation, tachycardia, thyroid hyperplasia, tremor and exophthalmos. The more important of these were: periods of pyrexia for which no assignable cause could be found; very low systolic blood pressure; purpura with prolonged bleeding time; decreased platelet count; swollen and bleeding gums and a history of profuse menstrual hemorrhage and a rapid gain in weight and muscular strength, rise in blood pressure and decrease in bleeding time associated with administration of fresh ox suprarenal gland, but without any noteworthy changes in the pulse rate, exophthalmos or thyroid gland. Very rapid and striking improvement in the general nutrition occurred during the administration of fresh ox suprarenal cortex, in 5 gm. daily doses by mouth rather than during the administration of desiccated suprarenal gland. The observation suggests a possible relative functional insufficiency of the suprarenal cortex as one of the underlying factors in exophthalmic goiter. Larger doses, especially of whole fresh suprarenal gland, caused nausea and vomiting, probably from direct irritation of the gastric mucosa by epinephrin. Evidence, both experimental and clinical, is now rapidly accumulating that the suprarenal gland, and particularly its cortical portion, plays an essential and fundamental rôle in the etiology of exophthalmic goiter.

Victor, M. ACUTE SUPRARENAL INSUFFICIENCY IN INFANCY. [*Zeitschrift für Kinderheilkunde*. August 19, 1921, XXX, No. 1-2.]

Severe symptoms of collapse in fourteen months old child suggested poisoning, but necropsy showed extensive destruction of the suprarenals. A second case, a seven months male infant, showed similar findings. The author holds that inferior suprarenals had been injured during delivery. They had sufficed during the early months of life, but broke down as greater demands were made on them as the child grew, complicated possibly by infection. In the second case, an acute sepsis accompanied by hemorrhages in the suprarenals and skin was probable. The clinical signs were convulsions, soft irregular pulse and cyanosis, contracted pupils and stupor. In both infants the thymus was exceptionally large. The syndrome, the author suggests, might have been benefited by epinephrin.

Schnyder, K. APLASIA OF SUPRARENAL. [*Schweiz. medizin. Woch.*, July 1921, LI, No. 28.]

This pathological and clinical study reviews the literatures in which are recorded six cases in which the right suprarenal capsule was missing, in two of which Addison's disease developed as the other suprarenal developed tuberculosis. The author here reports a third case of aplasia of the right suprarenal in a man of 68, with cancer of the larynx. Bronzing of the skin and brown spots in the buccal mucosa were present, but the blood pressure was not low. Twenty-four years previously he had been treated for scurvy, and "plus Addison's disease" is

noted in his early history card. Congenital absence of the right suprarenal was revealed at autopsy but the single one found seemed to be normal.

Brösamlen. EPINEPHRIN HYPERGLYCEMIA. [Deutsches Archiv. für klin. Med., October 21, 1921, CXXXVII, No. 5-6.]

Subcutaneous injection of epinephrin in healthy persons is known to bring about a hyperglycemia, reaching its maximum usually in about an hour. In this series of observations sugar in the urine was observed in four of thirty-five tests only and these four showed evidences of exophthalmic goiter, advanced tuberculosis, obesity or leukemia.

Girou, J. HYPOTENSIVE ACTION OF EPINEPHRIN. [Paris Médicale, October 1921, XI, No. 43.]

This article contains a critical review calling for reconsideration of present day interpretations of epinephrin action. In more than 100 test injections of 1 mg. epinephrin, given to stimulate the weakening heart, the blood pressure dropped in 80 per cent. Larger doses are given every day, although Kircheim's 48 mg. a day is still deemed hazardous. He cites some American experiments which tend to sustain his statement that small does lower and large doses raise the blood pressure.

Tronconi, S. HEMORRHAGE IN THE SUPRARENALS IN THE NEW BORN. [Pediatria, March 1921, XXIX, No. 6.]

This infant died the fifth day after birth. Intense jaundice was the most striking objective finding, but on autopsy hemorrhages were found in both suprarenals. There was a hematoma in one which was so large that it caused pressure on the bile duct; thus causing a purely mechanical jaundice. The suprarenals had evidently been diseased, and the hemorrhages were brought on, the writer thinks, in the act of swinging the child (Schultze). The mother also died of exhaustion, suggesting maternal suprarenal inferiority.

Tatum, I. A. L. EPINEPHRIN HYPERGLYCEMIA. [Journ. of Pharmacology and Experimental Therapeutics, June 1921, XVII, No. 5.]

According to this study the real mechanism of epinephrin mobilization of carbohydrates is as yet undetermined.

Kay and Brock. WHITE ADRENAL LINE. [Am. J. of Med. Sciences, April 1921, CLXI, No. 4.]

In this clinical study the authors come to the conclusion that the so-called "white line" of Sergent is a local vasomotor reflex, resident in the skin, bearing no direct relationship to suprarenal gland activity. They examined 255 individuals, many healthy and many sick. They find that the line appears or is absent: (a) Independent of blood pressure, acute fatigue and other signs of hypo-adrenia; (b) its frequent occurrence in normals and in a variety of diseases unassociated with hypo-adre-

nia; (c) its appearance in the fase of persistent general manifestations of epinephrin subcutaneously administered; (d) its peculiar association with scarlet fever. It would appear that the state of the vasomotor system which allows of its best exhibition is found in young adults of either sex, and especially in the exanthem of scarlet fever. This "white line" cannot be held to have the specific characters attributed to it by Sergent.

Exner, H. V. FUNCTIONS OF SUPRARENAL GLANDS IN WHITE RATS. [Dublin Journ. of Medical Science. J. A. M. A.]

Exner is convinced by the results of his extensive experimental work that the glycogenic function of the suprarenals is dependent on, or works in conjunction with, some nervous control; this nervous control, when adequately stimulated, still being able to produce glycosuria independently, and in the absence of all suprarenal tissue. Whether the converse is true is a matter for speculation, but whatever the glycogenic function of the suprarenal glands may be, it seems to be subordinate to and dependent on the central nervous system.

Peabody, Sturgis, Tompkins, Wearn. EPINEPHRIN HYPERSENSITIVENESS AND HYPERTHYROIDISM. [Am. J. of Med. Sciences, April 1921, CLXI, No. 4. J. A. M. A.]

Summarizing the observations made on various groups of subjects without evidence of organic disease, Peabody *et al*, state that hypersensitiveness to epinephrin is certainly not characteristic of the hardened soldier; that it occurs in about 14 per cent of average young men, such as medical students; that it is present in nearly 50 per cent of the type of young men who broke down under military training with the picture of "effort syndrome" and that it is still more common among definite psychoneurotics. It is difficult not to see some relation between epinephrin hypersensitiveness and what one may call a "nervous constitution." Different individuals, both sick and well, react with different degrees of intensity to the injection of epinephrin. The fundamental nature of the reaction is unknown. Hypersensitiveness to epinephrin is found in many patients with the clinical picture of hyperthyroidism and with an increased basal metabolism, but it is not constant under these conditions. The "positive" reaction to epinephrin appears to occur most often in highly nervous individuals, but it is not constant in such persons. The clinical significance of the reaction is not clear, but at present it should certainly not be regarded as having any specific significance in the diagnosis of hyperthyroidism.

Figenschau, K. J., and Berner, J. H. ADDISON'S DISEASE IN CHILD. [Norsk Mag. for Laegevidenskaben, March 1921.]

This is a clinical study of a girl of four who had Addison's disease. Necropsy showed that the left suprarenal gland was transformed into a ganglioneuroma with some small metastatic tumors. The clinical history

led to a diagnosis of mesenteric tuberculosis. The abdomen was distended but no ascites was found. The tumor weighed 50 gm. Forty ganglioneuroma are collected from the literature, including seventeen in children. In all, the tumor had developed from the sympathetic nervous system and the suprarenal. In another group of thirteen cases, including one child, the ganglioneuroma had developed in the central nervous system.

Lutembacher, R. ACTION OF EPINEPHRIN IN HEART BLOCK. [*Presse Méd.*, February 12, 1921.]

This clinical experimental study throws some light on the effect of epinephrin administration on the heart with total and partial heart block. There is an acceleration of the beat of the auricles and of the ventricles independent one of the other. The consequences are liable to be disastrous. The stimulating effect on the heart is evident, but its effect on conductivity may be slight or nil. Intravenous injection of epinephrin while accelerating the ventricle beat often entails long pauses followed by syncope. The most dangerous feature of the epinephrin treatment, however, is that after the pause of acceleration the heart beat may be seriously retarded for several days. This inhibition may be dangerous in case of complete dissociation of the auricle and ventricle.

Carter, W. E. ADRENAL MALIGNANCY. [*Am. Jl. Dis. Children*, 1921, XXII, No. 3.]

This pathological condition is held to be not infrequent in this general paper. He adds three cases of his own. An orbital hemorrhage is the first sign observed, and it may occur before any tumor is palpable. This is the more usual finding. The orbit involved is usually on the side of the tumor. Diagnosis is not difficult once the orbital hemorrhage has occurred; the disease is likely to be mistaken only for trauma, chloroma and scurvy. Surgical interference is of little service, except as a palliative to drain a pyonephrosis or to meet other complications, as the metastases usually occur before a diagnosis can be made. Metastases probably occur through the lymph stream. The medulla of the suprarenal being neuroectodermal, these tumors are similar to malignant neoplasms of the sympathetic nervous system, hence described as neuroblastoma.

Tokumitsu, Y. SUPRARENAL CORTEX. [*Mitteilungen a. d. Path. Inst. Univ. Tokio*, May 1921, I, No. 2. *J. A. M. A.*]

Tokumitsu describes what he calls a new function of the suprarenal cortex, which becomes manifest when a ligature is thrown around the pancreatic duct. The cortex proliferates and hypertrophies, evidently as a compensating process. The medulla of the suprarenal, on the other hand, seems to have an antagonistic action to that of the pancreas. The medulla and the cortex are separate organs. His research has confirmed that diabetes develops even with slight changes in the pancreas

if the suprarenal cortex shows degenerative changes, while otherwise the diabetes develops only with pronounced changes in the pancreas.

Webb, G. B., Gilbert, G. B., and Ryder, C. T. ENLARGEMENT OF ADRENALS AND THYROID IN TUBERCULOSIS. [Am. Rev. Tuberc., May 1921, 266.]

Webb, Gilbert, and Ryder have made observations on the weight of the adrenals in guinea pigs in health and in various diseases. In experimental tuberculosis they find a distinct increase, both absolute and relative to body weight. This becomes evident, on an average, about six weeks after subcutaneous inoculation, and increases with the generalization and progress of the disease. The possibility of error due to emaciation has been carefully excluded. The enlargement appears to be limited to the cortex. In only one animal were there tuberculous lesions in the adrenals. These were in the medulla. An even more pronounced enlargement was observed in guinea pigs suffering from pyogenic infections. The authors refer to a previous report in which thyroid enlargement was noted in experimental tuberculosis, and call attention to the frequent swelling of the thyroid in an early human tuberculosis. In conclusion they say, "This enlargement of adrenals and thyroid is probably in response to a demand for increased function, and it may be desirable to supplement this tendency by giving adrenal and thyroid extracts in selected cases of human tuberculosis." [Author's Abstract.]

Webb, Gilbert, and Ryder. ADRENALS AND THYROID IN TUBERCULOSIS. [Tr. Am. Phys., 1921.]

In a somewhat later paper Webb, Gilbert, and Ryder add the fact that some of the highest adrenal weights noted have been in guinea pigs dying of acute epizootic pneumonia, and call attention to the correspondence between their experimental results and T. R. Elliott's observations on human autopsy material, both in tuberculosis and acute pneumonia. They discuss the relation between hyperthyroidism and tuberculosis, including the difficulties in differential diagnosis, which are increased by the fact that tuberculosis may not only stimulate but excite a degree of hyperthyroidism. They cite the fact that marked hyperthyroidism appears to protect against tuberculosis, and comment on the mononuclear leucocytosis which has been observed in exophthalmic goiter, and which they find quite constantly in cases of tuberculosis which are doing well. They state positively that "in general, those patients whose thyroids show slight palpable enlargement do better than those with small inactive glands." [Author's Abstract.]

Achard, Ribot, and Binet. EPINEPHRIN HYPERGLYCEMIA. [Rev. d. Méd., September-October 1921, XXXVIII, No. 9-10.]

This is an experimental study on the action of epinephrin on the sugar content of the blood and the relations between epinephrin and

pancreas extract in respect to the storing and combustion of sugar in this line in animals—dogs. Injection of epinephrin, after pancreas removal, did not increase the sugar content of the blood. Otherwise, epinephrin seemed to check the sugar holding and sugar oxidizing properties of the dogs.

Kieley. JAMES' THEORY OF EMOTIONS IN RELATION TO SUPRARENAL GLANDS. [Journ. of Lab. and Clin. Med., January 1921, VI, No. 4.]

This clinical experimental effort was conducted upon two patients having a "fear syndrome," falsely labeled "psychasthenics." "Fear of death" was prominent in one of the patients. Kieley gave 1 to 2 c.c. of 1 per cent apocodeine solution, every other day, until twenty injections were reached. No effect on her anxiety was obtained. The second subject, during the influenza epidemic, had been advised to drink whiskey for prophylaxis. He had one or two drinks a day for three months and dates his fears from that time. His phobia was mainly that of committing suicide. He was contented with life but was obsessed by a fear of self-destruction. He received fifteen injections of 1 per cent apocodeine solution, ranging in dose from 1 to 2 c.c. There was no relief from the fear. "An emotion can be experienced independently of the physical changes which habitually accompany it" is the large conclusion drawn from this fragmentary study.

Friedman. POSSIBLE RELATION OF SUPRARENAL CORTEX TO EXOPHTHALMIC GOITER AND TO MYXEDEMA. [Med. Rec., February 5, 1921, XCIX, No. 6.]

In this general review the author first discusses the anatomy and physiology of the suprarenal gland. The separate functions of the cortex and the medulla are indicated. Not a single case is on record, he states, in which life was possible without a cortex. Some individuals, on the contrary, can survive without the medulla. That the cortex and the medulla are united in man and higher animals, and not separated as in many lower vertebrates seems to indicate some close functional integration. The chief function of the cortex is to supply the medulla, directly or indirectly, with products for the elaboration of the cortex-adrenalin. A second function of the cortex may be detoxicating. Control of body weight and of blood pressure are related to the cortex. It regulates the purin metabolism of the body and the physiological pigment metabolism. It probably destroys pathogenic bacteria of the body. Some of the clinical symptoms found in individuals afflicted with Addison's disease, such as emaciation, asthenia, and hyperpigmentation, manifest themselves to a lesser degree in individuals suffering from exophthalmic goiter. The symptoms have been shown to be in some way related to various degrees of inferiority. The gain in weight and the absence of true asthenia in myxedema may be due indirectly to hyperfunction of the cortex.

Claude, Henri. INCOMPLETE FORMS OF SUPRARENAL VIRILISM (WOLFFIAN VIRILISM). [L'Encéphale, November 1921.]

Besides suprarenal virilism consisting of a congenital state of pseudohermaphroditism with presence of a suprarenal tumor of Wolffian origin there are forms less pure, acquired, represented by transformation of the secondary sexual characters of the feminine to the masculine type. They are found not only among elderly women but in young subjects who, to morphologic, somatic troubles, have added also mental disturbances more or less characteristic. I have observed a very large number of cases of virilism which term incomplete forms (former fruster) because on the one hand the psychic and psychosexual difficulties were slightly pronounced and because the congenital glandular troubles were little apparent and could be assumed only on biological investigation. It seems that this dystrophy has been very specially realized in the ovarian neoformations of the luteinic type with secondary suprarenal reaction of the rather neoplastic type. The characteristic element would be merely the multiplication in the ovary of the luteinic cell under the metatypical form. But this luteinic cell is an element of Wolffian origin like the cortico-suprarenal cell. It is possible that the disturbances in the functional activity of this cell, which is the active element of the ovarian follicle as of the interstitial gland of the ovary, would form the origin of the dystrophy which constitutes the virilism. Hence it deserves the name of *Wolffian* rather than of *suprarenal virilism*. [Author's Abstract.]

Béclère, A. OVARIAN STERILIZATION AT ONE EXPOSURE BY THE HELP OF THE X-RAYS. [J. de radiol. et d'électrol., 1921, V, 67.]

The critical review of Béclère traces the development of the X-ray treatment of fibromyomata from its inception by Foveau de Courmelles in 1904 to the present-day methods advocated by the late Professor Krönig and continued by Seitz and Wintz. The chief part of the review is devoted to a critical examination of the efficacy and desirability of the German method, which consists in giving at one séance a dose of radiation which is calculated to give the desired results. From a careful examination of the publications which have issued from the Fribourg and Erlangen clinics, Béclère finds a not unimportant difference in the dose which is administered at the two institutions, for what must be considered the same object. Krönig and Friedrich estimate what they call the ovarialdosis to be 20 per cent of the erythemdosis, whereas the Kastrationdosis of Seitz and Wintz is 34 per cent of this same quantity. This difference may possibly be referable to an inequality of the standard employed at the two centers, namely, the erythema dose. The main questions raised by Béclère are two. First, can the changes in the ovary be brought about by a single application of X-rays with certainty. From an examination of published results he states that this cannot be done, and that it may therefore be necessary to repeat the exposure. The

second question centers around the desirability of such single exposure methods. The economical advantage is, in his opinion, counter-balanced by the ill effects upon the patients which frequently supervene upon these prolonged exposures to X-rays, and which are not often met in the conservative method of spacing the irradiations over a considerable length of time. Bécèle also discusses in some detail the arguments which have centered around the question as to whether the diminution in size of a fibroma is a sequel to the changes initiated in the ovary or whether it is due to a direct action of the rays upon the growth. The argument is well considered and the paper is a valuable one. It contains a useful bibliography, in which we fail to find the name of a single British author. [Medical Science.]

Fuchs, H. THE TERMINAL PHENOMENA AFTER MENOPAUSE INDUCED BY X-RAYS. [Strahlentherapie, 1921, XII, 742.]

On the basis of 69 cases the author compares the terminal condition of women in whom an artificial menopause has been produced according as that condition has followed on X-radiation, ovariectomy, or hysterectomy. The paper is entirely clinical and goes into some detail. He claims that under X-rays psychical equilibrium is better preserved, vasomotor changes are fewer, and there is less disturbance of the entire generative system. [Medical Science.]

Mahnert, A. ENDOCRINE GLANDS DURING PREGNANCY. [Archiv. für Gynaekologie, Berlin, 1920.]

This is an Abderhalden technic study in which the author finds that there was specific digestion of hypophysis albumin in 60 per cent of the twenty-five pregnant women. These frequent positive findings seem to indicate dysfunction of this gland at this time. Similar findings and conclusions are reported for the pineal gland in 40 per cent of twenty women, while 75 and 70 per cent were the proportions in twelve for the ovaries and suprarenals, and the thyroid 57 per cent. These glands and other organs are evidently undergoing marked variations in function which he calls "abnormal." The disturbances and dysfunction may be due to substances generated in the placenta. The endocrines may be constitutionally inferior, and thus unable to stand the stress of the pregnancy. The pineal seems to retrogress during a pregnancy according to the Abderhalden criteria.

Hutinel, V., and Maillet, M. GLANDULAR DYSTROPHIES AND PARTICULARLY MONOSYMPTOMATIC DYSTROPHIES. [Annales de Med., 1920, X, Nos. 2, 3, 5, 6.]

The endocrinous dystrophies can present themselves in all degrees. Sometimes they are frustrated and incomplete, sometimes it is difficult to determine their origin.

(a) The most striking and those earliest recognized are the *total cachexias*. They attack all the organs, all the tissues, following sup-

pression of a necessary function. Myxedema is the type best known; it opposes itself to the dysthyroidism of Basedow's disease. In the dystrophies of suprarenal origin Addison's disease may in the same way be opposed to the suprarenal-vascular syndrome of hyperadremia.

(b) The most frequent dystrophies are certain ones less profound, generally considered as the result of a lesser disturbance of the glandular activity. Instead of being total they attack preferably certain anatomic systems and are thus more *systematized*. They are as concerns the thyroid, the hypothyroidisms, the Basedowian syndromes and thyroid instability. In regard to the hypophysis they are acromegaly, gigantism, hypophysial nanism, the adipose-genital syndrome and the dystrophy of adolescents. Among the suprarenal syndromes of this category we must place frustrated Addisonianism, slight suprarenal insufficiency, pseudo-hermaphroditism, virilism and suprarenal nanism. The influence of the genital glands upon nutrition is considerable as one may observe among castrates. These disorders and those which may be imputed to the other glands have a particular importance at the time of growth and adolescence, which are always impeded. From this arises the various infantilisms, thyroid, hypophysial, suprarenal, renal, without including Lorain's type. One may recognize the importance in infancy of the study of the glandular synergies. These systematized dystrophies may attack the skin and notably the hairy system which develops badly in those with hypothyroidism, so much so that with certain changes of the suprarenals or of the ovaries one perceives a veritable hirsutism. Myxedematous infiltration, increase of fat and scleroderma are three forms of dystrophy which one meets among those with hypothyroidism. The circulatory apparatus suffers indirectly from the disturbances of the hypophysis and the suprarenals. The composition of the urine is often modified by the endocrinous disturbances (polyuria, diabetes). In the same way one recognizes the effect of internal secretions upon the nervous system, notably upon the sympathetic and the pneumogastric. The bony marrow is particularly sensitive to their action (rachitis, osteomalacia). Finally the glandular insufficiencies alter the resistance of subjects to the infections and the intoxications. In a number of the constitutional states one finds the mark of certain endocrinous insufficiencies.

(c) The *monosymptomatic* dystrophies apparently affect only one organ or a segment of an organ. They become often more profound as they attain their localization. They may arise from other causes than the endocrinous lesions. They are divided into two groups. In the first there are found arrests of development or the *aplasias*, which it is interesting to compare with the *atrophic* regressions of the same organs. In the second group nutrition is carried on in an abnormal manner. In the first nutrition is arrested or undergoes regression, in the second, it takes an injurious form. The hypoplasias are met with at each step in the history of the glandular affections. The aplasias, variably following the phases of existence in which they appeared,

present themselves under three types. In the most serious forms they are *congenital*; in the more common form they are *evolutive*; in the more retarded forms they are *regressive*. The authors study successively the aplasias of the genital organs, testicles and ovaries; those of the kidneys which may occasion true cachexias analogous to the endocrinous cachexias; those of the interstitial nephritides, of the chronic albuminurias and of the intermittent albuminurias; those of the nerve centers, on the one hand the congenital ones, mongolism, myotonia congenitale, etc., on the other hand the evolutive ones, particularly dementia precox; those of the circulatory system, notably chlorosis; the bony dysplasias, achondroplasia, periosteal dysplasia, congenital rachitis, etc., and the total dysplasias.

They devote the third chapter to the study of certain local disorders of nutrition which lead one to think of the endocrinous disturbances although they may be due to other causes. These disorders are circumscribed *edemas*, local *adiposities*, partial *sclerodermas*, local circulatory disorders, cutaneous, nervous, urinary, respiratory reactions. They close with general considerations upon the relation of the *diatheses* and of morbid temperaments with the endocrinous glands. [Authors' Abstract.]

Frey, W., u. Hageman, E. ADRENALIN LYMPHOCYTOSIS AS A TEST FOR SPLENIC FUNCTION. [Ztschr. f. klin. Med., 1921, XCII, 450. Med. Sc.]

It has been shown that 20 minutes after an injection of 1 mm. adrenalin into man a lymphocytosis takes place in which the number of these cells may be as high as 2,500. This appears to be due to a mechanical mobilization of lymphocytic elements in the spleen. The authors have attempted to ascertain whether pathological conditions of the spleen can be detected by the absence of this phenomenon. Sixteen cases of splenomegaly were investigated before operation, and the condition of the spleen ascertained from the histological picture. In only one case did the adrenalin test give a doubtful result. A negative reaction to the test indicates a severe pathological condition of the splenic tissue. The degree of lymphocytosis is dependent upon splenic function. A negative test is given when the number of leucocytes is decreased to less than 1,500 twenty minutes after injection.

Lobstein, J. A CASE OF EUNUCHOID GIANT-GROWTH. [Nederlandsch Tijdschr. voor Geneeskunde, 1922, LXVI, 743.]

The patient was a man of forty-four; as a child he was imbecile, late in talking and walking, showed intellectual and moral defects, could not learn a trade, and had enuresis for a long time. For twenty years he was in asylums: in course of time became more tractable. In 1920 he showed tall stature, kyphosis, exceeding long arms and legs, a small skull with low forehead, a pale skin, adiposity of lower abdomen and

nates, a boy's voice, very small penis and testes, no sexual libido, and polyuria. Possibly he had some limitation of his visual fields, but he had high myopia and drooping upper eyelids so that there was some doubt about the limitation. There was no reason to assume the existence of a lesion of the floor of the third ventricle to explain the polyuria. [Leonard J. Kidd, London, England.]

Mouriquand, G. OBESITY IN CHILDREN. [Bull. Méd., December 17, 1921, XXXV, No. 51. J. A. M. A.]

Mouriquand remarks that treatment of obesity in children should begin before they are born, before conception, the parents taking a course of physical treatment to combat their obesity, as well as dieting. The pregnant woman should refrain from fattening foods, but should not starve herself. Weill has reported the case of an obese woman who fairly starved herself during her second pregnancy, but this second child was even larger than her very large first child. Overfeeding of the infant must be guarded against with special care in families predisposed to obesity, and at weaning and later the more fattening elements must not be allowed in excess. It is sometimes advisable to get the child out of an environment with sedentary and overeating habits. Some children eat an enormous amount of bread; by reducing this, the tendency to obesity may be averted. Respiratory exercises increase the chest measure and oxidations, and training the muscles improves the "draft." With any tendency to myxedema, thyroid treatment may be effectual, but under other conditions it should be given very cautiously. It may increase the appetite, and thus do harm. When an endocrine origin is suspected, a combined organotherapy might be tried, alternating thyroid and ovarian treatment for a girl at puberty, or a combination of pituitary, ovary, and suprarenal extract with a minute amount of thyroid. This associated organotherapy has to be kept up for a long time, with intervals of from two to four weeks.

Jacoby. ENDOCRINE ASPECT OF FEMALE STERILITY. [Med. Rec., February 11, 1922, CI, No. 6.]

This general article discusses the problem of sterility. The chief cause is laid at the door of the ovary. Careful physical examination of the individual will usually reveal the gland or glands responsible for or participating in the ovarian inferiority. The pituitary, thyroid and suprarenals are chiefly implicated. The dysfunction of one or the other or several of these glands breaks up the pregnancy chain. The use of the proper gland extracts can frequently aid the dysfunctioning gland. Attention is called to a large class of cases which heretofore were either dilated or curetted or subjected to mutilating operations, or else were put off with placebos and hopes. A careful endocrine status is desirable then in all types of sterility not founded in gross objective pathological disturbances.

Carbus and O'Connor. FAMILIAL OCCURRENCE OF UNDESCENDED TESTES. [Surg., Gyn. and Obs., February 1922, XXXIV, No. 2.]

This casuistic contribution to heredity cites the cases of six brothers with testicular anomalies. Father and mother were not obviously involved. One sister is normal; a second sister gives evidence of gonadal disturbances.

Lydston. IMPLANTATION OF THE SEXUAL GLANDS. [Arch. Med. Belges, November 1920.]

Lydston contributes an article on this subject and complains that he has been ignored by both Voronoff and Steinach. His own début in the literature of this subject goes back to 1914. In one influential journal alone he published during that year no less than seven communications, and it is difficult to comprehend how his priority could have been ignored in Europe. The author then abandons the question of credit due and sums up the results of his extensive experiences, including one personal one. The recipient of the graft obtains a certain amount of hormone from the gland itself. Then the implanted interstitial cells of Leydig continue to produce hormone for a longer or shorter time or until the implant has been absorbed or has come away. But the recipient obtains something in addition to the hormone, and this is probably an enzyme or other principle which acts as a stimulant to the entire organism. In other words, the hormone which produces the return of sexual potency may be distinct from that which generally stimulates the organism. In regard to human donors the glands may be derived from the victims of accident, acute poisoning, the drowned, gas-intoxicated, and those killed by hanging (but not the electrocuted). The criterion is instantaneous death, but this point could be stretched a little to include those who survive for a few hours.

Falcone. IMPLANTATION OF SEX GLANDS. [Rif. Med., December 18, 1920, XXXVI, No. 51.]

In this clinical experimental paper the results of implanting half of the testicle of a sheep in the abdomen of four men of fifty-three, sixty-three, sixty-nine and seventy-four are recorded. The grafts were cast off as foreign bodies in two or three weeks after the operation in all but one case. Here the graft seems to have taken. The implant acts as a form of organotherapy. The cytolytins which are assumed to be set at liberty exert power as a secretion, and the resulting stimulation seems to be permanent. Suggestion can be excluded, he thinks. The general action was beneficial and the testicles increased in size in all.

Movak, Emil. THE ENDOCRINE GLANDS IN CERTAIN MENSTRUAL DISORDERS. [Endocrinology, July-September, 1920.]

This general paper emphasizes the importance of vegetative nervous system in its regulation of the menstrual cycle. The explanation of its

mechanism is to be sought in a study of the functions of certain endocrine glands. Disorders of menstruation are often found with disorders of these glands. There is with menstruation some association with the cortex, as is shown by the occasional occurrence of menstrual aberrations under the influence of symbolic activities. Ovarian influence is exerted through the blood stream and not through the nerves, as menstruation occurs when the ovaries have been transplanted. [Not proof?] The weight of evidence is in favor of the view that it is the corpus luteum which plays the most important part in regard to the menstrual function. But the ovary probably produces more than one hormone, as it influences the development of the sexual characteristics and the general body functions. The internal secretion of the ovaries is closely interrelated with that of other endocrine glands, as disease of the latter influences menstruation. Amenorrhea associated with obesity is generally recognized as being due to hypopituitarism. Primary or spasmodic dysmenorrhea is most commonly caused by hypoplasia or defective development of the uterus. The hypoplasia may be classified under three heads: (1) In the fetal type the body is extremely rudimentary; the cervix is comparatively large. (2) In the infantile type the body is not so rudimentary and there is often an associated antelexion. (3) In the subpubescent type hypoplasia is relatively slight. This want of development of the body of the uterus cannot be due to secretion from the corpus luteum, as it does not form till after puberty. There is reason for belief that the earlier development of the uterus is under the influence of other endocrine glands and especially the hypophysis. Functional uterine bleeding is extremely common, especially at the two extremes of menstrual life. Most commonly it takes the form of menorrhagia rather than metorrhagia and careful examination may show normal pelvic organs. In a very large proportion the endometrium conforms to the type described by Cullen as hyperplasia of the endometrium. This type of endometrium is found in association with uterine bleeding. The reverse, of course, is not true, for uterine hemorrhage is due to many anatomical causes. There is good reason to believe that this characteristic change in the endometrium is due to disturbed function of the ovary and most probably due to hypersecretion of that element which is concerned with the production of normal menstruation.

Stühmer and Dreyer, K. SEROLOGIC TESTS IN PREGNANCY. [Zeits. u. Geburt. u. Gynäk., November 12, 1921, LXXXIV, No. 2. J. A. M. A.]

Stühmer and Dreyer applied parallel Wassermann and other serologic tests twice a week in their maternity cases, and they thus have a series of 1,000 tests of parturients' serum, with 2,500 control tests of known syphilitic or nonsyphilitic serum. These experiences show that the serum of healthy women may respond positively to the Wassermann test during pregnancy and childbirth. Unreliable findings were obtained in

fully 10 per cent. The retroplacental blood is especially liable to respond misleadingly, and also the blood in the umbilical vein. The fewest erroneous responses were obtained with the Sachs-Georgi flocculation test.

Gigou. DWARF AND GIANT GROWTH. [Schweiz. Arch. f. Neur. u. Psych., 1921, IX, No. 2.]

This clinical paper tests out the secretory functions in relation to growth. Eight individuals, dwarfs or giants, are studied. They include a man of twenty-one only 87 cm. tall; a girl of four, 73.5 cm. tall; a woman of twenty-two whose height is 207 cm., and a man of twenty-nine who, reclining, measures 237 cm., nearly 7 feet, 11 inches, the London giant known as "Bobs." His height, standing, is 234 cm. but he weighs only 112 pounds, this light weight being related to an osteoporosis evident in X-ray pictures. He is a true acromegalic type. The female giant was well proportioned and apparently healthy, but the menses were irregular, a slight tendency to eunuchoid type was present, and one exostosis on the right tibia gave evidence of the bony malformation of possibly pituitary origin.

Hirst, B. C. GLANDULAR EXTRACTS IN MENSTRUAL DISORDERS. [New York Med. Journ., October 5, 1921.]

This is a somewhat optimistic clinical paper in which the comparative values of whole ovarian extract, corpus luteum extract, and ovarian residue in menstrual disorders are set forth. The whole ovary extract is most useful in the natural surgical menopause in the late establishment of menstruation and in relieving the nervous and mental disturbances. The most satisfactory results occur in cases of the natural menopause, and the least satisfactory are those in early surgical menopause. Ovarian residue extract appears to be of more service in late development of puberty, irregular menstruation at puberty, infantilism, menorrhagia, obesity and amenorrhea. Corpus luteum extract has controlled the nausea of pregnancy, habitual abortion without apparent cause, functional amenorrhea, sterility and pruritus vulvae. These extracts are, according to Hirst, best administered intravenously, though they are put up for administration by mouth. Results are variable, especially in the case of ovarian residue extract, and may be slow in development, the quickest being obtained from whole ovarian extract in the nausea of pregnancy, while the most discouraging results are in cases of obesity and amenorrhea.

Rotter, J. CAUSE OF STERILITY IN WOMEN. [Kans. Med. Soc. Journ., December 1921; XXI, No. 12.]

A clinical study of gonadal and other endocrinopathies as related to sterility. Three patients are reported on: (1) A case of primary sterility. This, on analysis, he believes to have been due to a hypofunction of the

posterior part of the pituitary and ovaries. (2) A case of acquired sterility. This is analyzed as a dysfunction of the ovarian, thyroid, and pituitary; and (3) an example of hyperactivity of part of the posterior pituitary gland and ovaries, related causally, he thinks, to the excessive trophic and stimulating influence on the endometrium, which disturbed the imbedding and retaining of the fecundated ovum. *Post hoc, ergo propterea hoc*. Treatment along the lines indicated brought three babies.

Labhardt, A. PHYSIOLOGY AND PATHOLOGY OF THE OVARIES. [Schw. med. Woch., May 6, 1920.]

This general paper emphasizes the important rôle of the gonads, not alone for the generative activities but for the whole body. Gynecology is receding more and more from the conception of localized pathologic conditions. Instead of trying to treat the local process, the patient as a whole has to be treated.

Halban, J. INCRETORY PROBLEMS IN GYNECOLOGY. [Münch. med. Woch., October 14, 1921, LXVIII, No. 41.]

The author in this general paper shows how the newer neuroendocrine data have upset nearly all of the cherished hypotheses of recent gynecology. It has long been taught that the ovary is the center of the genital apparatus of the female, since after ovariectomy, as well as after the natural cessation of ovarian function during the climacteric, an involution or atrophy of the whole genital system occurs. It was formerly supposed that trophic nerves extended from the ovary to the other sexual organs, and that the atrophy that followed ovariectomy was due to the division of these nerves. But of late it has been shown that if the ovaries are removed and subsequently reimplanted elsewhere, atrophy does not occur, although the nerves have been divided in exactly the same manner. [Vegetative nerves are never really divided.] This allows the hypothesis that the trophic influence of the ovary must be due to chemical substances, and that the ovary is a gland of internal secretion. By experiments on apes in whom menstruation occurs every four weeks, is retained if the extirpated ovaries are reimplanted in the body. If the transplanted ovaries were removed, menstruation ceased.

Waldeyer et al. INTERNAL SECRETION AND SEXUALITY. [Zeits. f. Urologie, 1921, XV, No. 3.]

The entire number of this journal is devoted to the report of the joint session of the Berlin Medical Society for Sexual Research and Eugenics and the Berlin Urologic Society. The anatomy of the eight true endocrine glands was detailed by Waldeyer; of the six with both internal and external secretion; the three that are suspected of an internal secretion but it has not yet been demonstrated (mammary and salivary glands) and the nonglandular bodies to which an internal secretion is credited (spleen, choroid, plexus, myometrial cells, pyrrhol cells,

fat bodies, placenta and fetus). Richter pointed out that castration of males entails an infantile, asexual further development. Also that there are genital zones in the brain, especially on the floor of the third ventricle. The internal secretion is an important factor in the psychosexual sphere, but is not the only one. The endocrine hormones influence the brain, but the brain has also an important influence on internal secretion. In Steinach's experimental modification of the sexual characters, he added, only the sexual characters were modified; the sex itself was never altered. Richter commented on his microscopic findings in the testicles of pseudohermaphrodites, the homosexual, dwarfs, etc., remarking in conclusion that there is no conclusive evidence to date that the interstitial cells of the testicles are important from the morphologic standpoint. [J. A. M. A.]

II. SENSORI-MOTOR NEUROLOGY.

2. SPINAL CORD.

Castex and del Pont. CASE OF RECKLINGHAUSEN'S DISEASE. [Rev. d. l. Méd. Argentina, July-September 1920, XXXIII, No. 189-191. J. A. M. A.]

Castex and del Pont give a colored plate and photomicrograms of an extreme case of subcutaneous tumors of the Recklinghausen type in a man of forty-eight. The tumors show a sarcomatous structure with a tendency to fatty degeneration, but they were small and had never been noticed by the man who had applied for treatment for an acute throat disturbance. He presented numerous manifestations of tertiary syphilis, with hypertrophy of the heart and a gummatous bone process.

Stewart, M. J. MALIGNANT SACROCOCCYGEAL CHORDOMA. [(B.) J. Path. and Bacteriol., 1922, XXV, 40. Med. Sc.]

A well-reported and illustrated case of the rare tumor known as malignant chordoma (Ribbert) which arises from remnants of notochord and is met chiefly near the spheno-occipital synchondrosis (clivus Blumenbachii) and in the sacrococcygeal region. The patient, a man aged sixty-five, had a slow-growing tumor over the coccyx, and it attained the size of an orange. Five years after excision it recurred in the left buttock, and three years later a mass appeared over the right scapula. These also grew slowly and, while the scapular mass remained discrete and encapsuled, the tumor in the left buttock caused great destruction of the femur and iliac blade on that side, death occurring 11 years after removal of the primary growth. The histological characters of the tumor and the physaliphorous cells are described. A complete bibliography of most of the known cases accompanies the paper.

Baker. COMPRESSION FRACTURES OF VERTEBRÆ. [Surg., Gyn. and Obstet., October 1920. B. M. J.]

In describing seven cases of compression fractures of the vertebral bodies with delayed symptoms—the so-called “Kümmel’s disease,” the characteristic history is one of injury to the back with more or less local pain and temporary incapacity. There is no injury to the cord, and the absence of positive neurological findings leads the medical attendant to put the possibility of vertebral fracture out of court. Sometimes the patient may be up and about within two weeks, and the true diagnosis may not be made for periods of from three months to two years later. The injury is usually a fall, and the site of the compression fracture most often is in one of the lower thoracic or lumbar vertebræ. Pain is apt to be persistent, and subsequent examination will, as a rule, reveal a distinct kyphosis, or local angulation of the spine, with muscle spasm. Careful radiography displays a compressed vertebra. Positive diagnosis can only be arrived at through the X-rays, and it is most important that these should be clear and sharp. The radiography of the spine is not yet as certain as the clinician might wish. Often a skiagram taken at the time of the injury has disclosed no spinal abnormality, whilst later plates show an incontestable fracture. It is not quite fair to blame that radiographer in all cases for this, for the bony changes probably develop later. In four of Baker’s seven cases the initial roentgenograms were negative for bony lesion, and no early treatment was instituted. The treatment is the same as for ordinary fracture of the spine. Late nervous changes must be very rare and generally due to callus formation.

van Schelven, Th. TRANSVERSE SPINAL CORD LESIONS. [Psychiatrische en Neurologische Bladen, 1920, Nos. 5–6, p. 384.]

The writer describes a new syndrome due to wound of the spinal cord behind the transverse median line (through the bases of the dorsal horns). These cases are rare, and are mostly due to a bullet wound of the spinal cord from the side. The resulting symptoms occur symmetrically on both sides of the body. The syndrome consists of (1) spastic paraparesis, (2) ataxia, (3) loss or great reduction of deep sensibility, with (4) intact superficial sensibility. The region of the cord involved is limited to the dorsal horns, dorsal columns, posterolateral bundles, crossed pyramidal tracts, and the lateral cerebellar tracts. Immediately after the bullet wound there is flaccid palsy with loss of reflexes. After weeks or months some voluntary power may return. Later there is spastic paralysis, if the lesion be above the lumbar cord. In favorable cases the patient may be able to walk after some months. There is loss of deep sensibility, and loss of tuning-fork sensibility: strong pressure is felt merely as touch. But superficial sensibility is normal or but very slightly affected. In the early period after the trauma, however, there is almost complete anesthesia caudally of the

lesion. This is to be regarded as a sequel of shock that for a time interferes with conduction in the cord. Usually this absolute anesthesia disappears, and there is left merely a disturbance of the deep sensibility of the joint-, bone-, and muscle-sense. When voluntary movements again become possible, disturbances of coördination appear, but these are often concealed by contractures. In many cases there is definite ataxia. The tendon-jerks disappear immediately after the injury, but they soon return and become increased. The cutaneous reflexes are at first very sluggish or absent; later they return, but remain difficult to obtain. The plantar responses are extensor, often very soon after the injury. Usually there is urinary retention and fecal incontinence, but they are not usually permanent. The ataxia and the deep sensibility disturbance are to be explained by a lesion of the lateral cerebellar tract and the dorsal columns, the spastic paraparesis by lesion of the crossed pyramidal path in the hinder part of the lateral columns. The superficial sensibility does not suffer, for the fasciculus anterolateralis ascendens anterior (touch) and the fasciculus anterolateralis ascendens posterior (pain- and temperature-sense) are intact. Behind the transverse middle line of the cord are situated the fiber-systems which are connected with peripheral end organs on the same side of the body, *viz.*, the uncrossed sensory and the crossed motor paths. In front of this line are the uncrossed motor and the crossed sensory systems, *viz.*, the fibers connected with the opposite half of the body (peripherally) and with the same half of the brain (centrally). This rule holds good also for the cerebellum. A lesion in front of this line gives symptoms on the opposite side; a lesion behind it gives symptoms on the same side. [Leonard J. Kidd, London, England.]

Roubier, C., and Brette, P. TUMOR OF SPINAL MENINGES. [Annales de Méd., January 1920.]

In this clinical paper is described the case of a woman forty-eight years of age who for six or seven months had pains in the neck and left arm. This was followed by a spastic paraplegia and a paresis of the smaller muscles of the hands, predominant on the left with slight atrophy. Pains were severe enough to require morphin. Trophic and sphincter disturbances then developed. Necropsy revealed a small subdural myxosarcoma, not adherent to cord or meninges, but difficult of access.

Meyer, Carl. SPINAL CORD TUMOR AND PREGNANCY. [Zentralb. f. Gyn., March 6, 1921.]

This is a clinical study. After a brief historical résumé in which Bogdanowitsch, who in 1913 reported a case of spontaneous labor in a woman suffering from complete paralysis of the body and lower limbs is commented on. Bogdanowitsch's patient, with thirteenth pregnancy, showed from the third month symptoms of a tumor in the cervical

portion of the cord. She was brought to hospital moribund, and preparations were made for Cæsarian. Labor began spontaneously, however, and the child was born without artificial interference. The patient died two days later from respiratory involvement. The case of Meyer's was a prim'para, aged twenty-three, who had been treated five years before for rheumatic affections with angina and bradycardia. In January, 1919, she began to complain of pains between the shoulders. Pruritus of the arms, chest, and back, together with weakness of the right knee developed later. Later pain was also felt in the right leg. These symptoms appeared to coincide with the beginning of pregnancy, during the course of which there developed spasms of the lower limbs, especially the right. Abdominal reflexes were absent; speech remained unaffected, and there were no eye symptoms. Multiple sclerosis was diagnosed, but later a tumor was suspected. The onset of labor was attended by severe vomiting. The expulsion of the child took place without cognizance other than a sensation of stretching in the right half of the vagina. Six weeks later she died, and a spindle cell sarcoma was found at the level of the upper dorsal and lower cervical vertebræ.

Climenko, H. DIAGNOSIS OF SPINAL CORD TUMORS. [Med. Record, May 29, 1920.]

In this clinical paper Climenko discusses various symptomatic features (chiefly pain) of value in the diagnosis of spinal cord tumors. In three instances reported on, pain was a common factor. In two extra-medullary tumors, the pain was an early symptom, whereas in the one intramedullary tumor, it developed later. In one case the pain was like paresthesia at the beginning, and later assumed a sharp, lancinating character. It began as a severe sharp, cutting pain, and continued in two other cases. Pain is a valuable guide when properly interpreted. In this interpretation the mental make up of the patient must be borne in mind. Watch the patient's facial expression when questions are put and answers given. The facial expression is particularly valuable in testing for tenderness. Climenko also calls attention to the lumbar puncture. When following this procedure in one case, the patient became suddenly more paralyzed and the Brown-Séquardian syndrome became pronounced. The tumor pressed more on the cord and produced the given syndrome, is his explanation. Such a syndrome should be considered as pathognomonic of spinal cord tumors when vertebral disease can be eliminated from consideration.

Lhermitte, J. RADIOTHERAPY IN SYRINGOMYELIA. [Paris Médical, October 1921, XI, No. 40.]

This observer has had a number of years experience in the use of X-ray and radium therapy in spinal cord lesions. In this, one of a number of contributions, Lhermitte emphasizes that in syringomyelia there is rarely any trace of a neoplasm in these cavities in the spinal

cord traceable to hydromyelia, softening and necrosis, or a hemorrhagic focus. There is one variety of syringomyelia, however, with proliferation of neuroglia around and remote from the cavity. This gliomatous type of syringomyelia is that which seems to be amenable to the roentgen rays. The subjective symptoms subside first, and the hands and legs become more tractable. Muscular atrophy is not modified, but its progress may be retarded or stopped entirely. Bone and joint disturbances, however, seem to be less profoundly modified than the muscles in the initial lesion, hence they get better sooner. One case of perforating ulcer on a finger rapidly improved under radiotherapy. In genital and bladder disturbances from syringomyelia the results were favorable. Radiotherapy induces regression of the lesions to a certain extent, not mere remissions in the course. This was confirmed at necropsy in a case treated by the author in 1914. The cavity that had been treated had scarcely any trace left of gliomatosis while in the non-treated regions gliomatous tissue was active. There are no means of differentiating the form of syringomyelia in a given case as yet except possibly by the results of radiotherapy. He makes an exposure from each side, 4 H units of 8 or 9 B penetrating power, with a 4 to 6 mm. filter, and repeats this every week.

Church, A. PARAPLEGIC MULTIPLE SCLEROSIS. [Journ. A. M. A., June 12, 1920.]

Answers to a questionnaire sent by Church to a limited number of American neurologists tended to show that these neurologists are seeing more cases of paraplegic multiple sclerosis than formerly, and that they now recognize the forms and aberrant types with a readiness that is somewhat of a recent acquisition. In the last three years, Church has seen thirty-two cases in private practice alone. He states that considering that the lesions of multiple sclerosis are extremely widespread in the brain and spinal cord, without symmetry or systematization, it is quite surprising that the original clinical conception should have prevailed so completely and persisted so long. In many cases, symptomatically at least, the brain seems quite exempt; in others the cord is not materially disturbed; but in all, sooner or later both are implicated. Contrary to early descriptions, sensory disturbances, both subjective and objective in character, are not at all infrequent in this type of multiple sclerosis. Hypesthesia and girdling sensations of segment level distribution of very definite and persistent character have more than once given rise, in conjunction with other paraplegic features, to a diagnosis of pressure on the cord. Among the signs of the disease, when affecting mainly or only the lower extremities, are the pathologic reflexes—ankle clonus, Babinski's toe sign, intensified knee jerks, and last and most important of all, one-sided, partial or complete abolition of the umbilical reflex. An interesting and instructive observation is furnished by the lack of uniformity with which the reflexes are modified on the

two sides. The sphincter control in the late stages is not rarely weakened, and actual incontinence may occur. To the lower level symptoms of the paraplegic type there are commonly added some of the cephalic manifestations of cerebral participation. Squints, double vision, marginal optic atrophy, irregularities of the visual field, cerebellar symptoms, intention tremor, speech defects, and even mental peculiarities are, some or all, in slight or marked degree, sooner or later observed. The condition, therefore, as in all other manifestations of multiple sclerosis, becomes progressively worse, and complete disability the ultima. Fortunately, the patients suffer little, aside from the growing feebleness and helplessness, to which they progressively adjust themselves, and the disease does not in itself appear to be fatal. The treatment of multiple sclerosis is a frequent source of disappointment.

Juarros, C. ARSPHENAMIN IN MULTIPLE SCLEROSIS. [Siglo. Med., December 18, 1920.]

In this clinical experimental therapeutic article the author discusses the use of arsenic preparations in the treatment of multiple sclerosis. The type of sclerosis is not carefully differentiated. In three cases marked improvement took place after the use of arsphenamine. Disseminated sclerosis he defines as a disease of inflammatory nature, the work of some virus in the blood stream. Injection of spinal fluid and blood from patients with multiple sclerosis has been known to cause nervous cerebromedullary disturbances in animals, and a spirochete has never been found in the blood of such animals, distinct from the spirochete of syphilis (Kuhn and Steiner). He dwells upon the question of remissions which, because they are known to have been from 2-10 years standing, renders the problem of estimating the value of a therapy very difficult of accurate estimation.

Schuster, G. MULTIPLE SCLEROSIS AND PARESIS. [Lancet, January 1921. Ed. Aust. Med. Jl.]

It is several years now since Moore and Noguchi did much to elucidate the histopathology of paresis by his discovery of the *Spirocheta pallida* in the cerebral cortex of patients who had succumbed to the disease. Levaditi, Marie and Bankowski added to this valuable discovery the proof that it was impossible with our present means of investigation to demonstrate the presence of the organism outside the cortex. They also pointed out that it was discoverable only in certain circumscribed areas of the grey matter. Further, the organism was only isolated when the patient had recently been suffering from paroxysms or acute exacerbations of the disease. Schuster has recently pointed out that in cases of paresis the spirochetes are congregated chiefly in the lower cortical strata. In many instances they were found in large numbers in the border zone of cortex and medulla, while the degenerative forms were present chiefly in the upper cortical layers. He also

demonstrated the presence of spirochetes in the cortex of patients who showed no clinical evidence of paroxysms of the disease. In one case of disseminated sclerosis he discovered fine spirochetes, indistinguishable from the *Spirocheta pallida*, in the upper strata of the cortex. Schuster draws a comparison between this disease and paresis. The patches of sclerosis in the former are frequently more or less confined to the corticomedullary boundary, which would appear to have some obscure microchemical affinity for the causative agent of both diseases. In disseminated sclerosis there is a plexiform disintegration of the myelin sheath of the nerve fibers and a similar histological change has been frequently observed in cases of paresis. Further, in the latter disease the disintegration occurs at the boundary of cortex and medulla. Lastly, a similar curve is obtained from Lange's colloidal gold reaction in disseminated sclerosis, general paralysis and cerebral syphilis. These observations may be found to be of importance in the elucidation of the etiology of multiple sclerosis. They are of further interest in focusing attention on that interesting anatomical zone which lies between the grey and white matter of the cerebrum.

Kalberlah, F. ETIOLOGY OF DISSEMINATED SCLEROSIS. [*Deutsche med. Wchnschr.*, 1921, XLVII, 102.]

Kalberlah inoculated rabbits intradurally and intraperitoneally with blood and cerebrospinal fluid from two active cases of disseminated sclerosis. One of the inoculated animals developed paralysis, and from its blood during life a spirochete was obtained. A second animal inoculated from this also developed paralysis, but its blood revealed no spirochete. The rabbit inoculated from the second case died within two weeks and the expressed liver juices, when stained by Giemsa's method, showed a spirochete similar to that found in the first case. The organism is plumper than *S. pallida*, and tapers to a point at each end. It has from three to six spirals. Neisser and Klein examined Kalberlah's preparations and confirmed his observations. Healthy rabbits and animals dying from other causes in the cages did not show a similar organism. Kalberlah names it *Spirochete polysclerotica*, and considers it to be in all probability the cause of disseminated sclerosis. [F. M. R. Walshe, *Med. Sc.*]

Steiner, G. MULTIPLE SCLEROSIS. [*Therapeutische Halbmonatshefte*, 1920.]

This is a popular contribution from Steiner who with Kühn made the initial statements relative to their new finding. In this paper the author only summarizes his findings. In rabbits and guinea-pigs he has succeeded in inoculating them with fluid and blood from recent cases of multiple sclerosis, and a spirochete was found in their tissues. A monkey inoculated at the same time developed paralysis eighteen months later, and the brain showed changes analogous in many respects to those found in multiple sclerosis in man.

3. PONS MEDULLA; MID-BRAIN.

Riley, Henry Alsop. THE SPINAL FORMS OF EPIDEMIC ENCEPHALITIS. [Am. Arch. Neurology, 1921, V, 408.]

This form was late of recognition being considered as an aberrant form of poliomyelitis associated with somnolence. Only the increasing number of cases made it evident that there was a distinct form of spinal involvement of epidemic encephalitis. Pure examples of any one form or type almost unknown, only the predominating picture determining the label to be applied to the disease.

Figures by Wechsler showed that out of 864 cases, 44 were described as myoclonic, 29 as radicular, neuritic or neuralgic, 24 as meningitic, 16 as myelitic or spastic, 5 as paraplegic, 2 as ventral poliomyelitic and 1 as dorsal poliomyelitic, a total of 121 cases. On account of the loose description and terminology, only 23 cases can be considered as spinal, the myelitic, paraplegic and ventral poliomyelitic.

The types of epidemic encephalitis which are apparently mainly spinal in character may be divided into two groups: (1) The ventral poliomyelitic type; (2) the transverse myelitic form. The ventral poliomyelitic form may be divided into two subgroups: (a) The irritative type; (b) the paralytic type.

Rarely seen as clear-cut entities, merging one into another; the paralytic form is often the end result of the irritative form. The irritative form may clear up without any permanent defect; the paralytic form may develop full blown. The literature is not clear in definite distinguishing features in motor disturbances, observations being mixed and conflicting. The type of movement is the criterion by which the level of the disturbance may be identified.

(a) The cortical level is distinguished by the movement being a purposeful synergized movement, presenting elements which distinguish not only the cortical pattern and concept, but also the collaboration of the cerebellum in the movement, the type characteristic of Jacksonian epilepsy. The arrangement of the Betz cells and the ventral horn cells makes the cortical type a synergized movement not the aimless contraction of independent muscles or muscle fibers. Such cortical stimuli are incapable of bringing about myoclonic or fibrillary contractions.

(b) The striate level is distinguished by the movements which we know as the choreas, the athetoses and the mobile spasms. The underlying feature being the automatic associated type of movement we can differentiate these from the spinal level of motility.

(c) The cerebellar level need not be considered for we do not have any definite type of abnormal involuntary movement from stimulation of the cerebellum.

(d) The spinal level of movement is the type that has characterized the myoclonic and the fibrillary manifestations of epidemic encephalitis.

litis. Such contractions at most produce a movement of a single muscle, at least but a single muscle fasciculus. They are simple, purposeless, irregular movements. The coarse myoclonic movements are twitching, exceedingly rapid in rate, irregular in rhythm and variable in extent. At times slow vermicular movements have been observed, probably due to stimulation of connector neurones in the spinal gray and the resulting correlated activity of several neurone groups. The myoclonic form was often preceded for several days by radicular pains; the interval may indicate the time necessary for the infective agent to spread from the roots to the parenchyma of the cord itself.

The paralytic form closely resembles that seen in poliomyelitis in fact of itself is indistinguishable from that disease, the history and concomitant phenomena making the differential diagnosis.

The transverse myelitic form is similar to that seen in other forms of spinal disease and varies with the site and extent of the lesion. By some it has been considered not as a definite type but as an accidental occurrence in the course of a typical spinal encephalitis, as a result of hemorrhage or thrombosis. Occasional xanthochromia may substantiate this view.

Case Reports. Irritative form from the Neurological Institute Service. Onset seven weeks before admission, pain and soreness spreading from head to entire body, retention of urine, transient diplopia, insomnia and tremor. Fixity of facial expression; coarse tremor of upper and lower extremities, transient cog-wheel in flexing forearms, brief jerky movements of toes and feet and myoclonic contractions of dorsal muscle groups in the thigh. Abdominal myoclonus. Reflexes were sluggish, abdominals absent, no pathological reflexes. No sensory changes of note. Slight external rectus weakness on the right side. Laboratory examination negative.

Myoclonic fibrillary type. Onset with pins and needles sensation, then pain in left instep, could not step, sharp, localized and stabbing. Spread to right leg. Subsided and followed by twitching movements in calves, thighs and abdomen. Was delirious, hallucinating with some expansive tendencies for some time. Could not walk on heels or toes, steppage, cerebellar function normal. Reflexes LKJ 3, RKJ 1; AJS 0. Abdominals R 2, L ?, no pathological reflexes. Muscle strength diminished in abductors of thighs both sides. Adductors of thighs left affected more than right. Extension and flexion of feet on leg very weak. Atrophy of left thigh and calf. Sensory: some change over right L5-S5; left L2-S5. Right pupil larger than left normal reactions.

Transverse myelitic form. Onset with dizziness, diplopia, headache, left facial paralysis. Right ptosis. Reflexes of upper extremity—active; of lower extremity—absent, no abdominal or pathological reflexes. No movement of lower extremity except slight flexion and extension of the

toes. Tone much reduced in lower extremities. Level at T2-4 with secondary level at S2 for all types of sensation. Slight papillitis in each eye, pupils equal, regular and central, both sluggish to light and accommodation, left more than right. Right ptosis, partial left ptosis, right eye immobile, left eye, deficient internal rectus. Partial left seventh. Straw colored spinal fluid, otherwise laboratory examinations were negative. As a result of the lumbar puncture the patient developed a total transverse lesion at the level of T2, complete paraplegia, retention of urine, pneumonia and death, by autopsy. (Author's abstract.)

Holden, W. A. OCULAR MANIFESTATIONS IN EPIDEMIC ENCEPHALITIS. [Arch. Ophthalmology, March 1921.]

W. A. Holden reports the following statistics of ocular manifestations in epidemic encephalitis. They are based upon the study of one hundred consecutive cases among patients at the Mount Sinai Hospital, New York. Blurring of the discs occurred in four cases, papilledema in one, ptosis in both eyes in 45, ptosis in one eye in 11, paralysis of one or both external recti muscles in 44 and nystagmus in 32. Irregularity of the pupils occurred in 15 cases, inequality in 20 and sluggishness or absence of light reflex in 35. Weakness of accommodation was present in both eyes in one case. There was a notable frequency in the association of ptosis and paralysis of the external rectus muscle—a combination rarely met with in any other disease. A curious condition sometimes present was paralysis of divergence without paralysis or spasm of the recti muscles and with normal ocular mobility. When converged on a point 15 cm. away the eyes could see single. Beyond this distance there was homonymous diplopia. The author has observed the condition in patients suffering from tumor of the midbrain and from uremia. Paresis of accommodation was observed without dilatation of the pupil.

Kraus, Walter M., and Pardee, Irving H. SEROLOGY OF THE SPINAL FLUID AND BLOOD IN EPIDEMIC ENCEPHALITIS.

The fluid is, in most cases, clear and colorless. Occasionally bloody fluid is obtained. As would be expected, the pressure of the spinal fluid is sometimes very much increased. Here also no rule exists, and, though this hypertension is frequent, it is not universal. On the average, more cells are found during the first three weeks of the disease. The value of this in individual cases is diminished by the variations in the number of cells in each week. The number of polymorphonuclear cells may be as great as 15 per cent. Fluctuation in the number of cells occurs during the course of the disease. When there is a remission the cell counts may increase. The globulin is usually increased to a moderate degree (in 72 per cent of the cases). However, since this is not always true, its diagnostic value in individual cases is lessened. The presence of globulin is not always paralleled by the presence of

cells or a positive colloidal gold curve. The Wassermann test was negative when reported. The colloidal gold curve has been found changed in 100 out of 120 cases (83 per cent). The type of curve varies considerably. There is a tendency toward elevation of the left-hand part of the curves in the later stage of the disease but that is not constant. Positive curves were found as late as the twenty-first month. Elevations of the right end of the curve alone were not found. The tendency is to a color change in the high and medium concentrations of spinal fluid. Changes in the low concentrations do not occur alone, but may occur when the medium and the high concentrations are altered.

The normal values for red blood count and hemoglobin have invariably been found. Leukocyte counts have varied from a minimum of 4,500 to a maximum of 32,000, the general average being 12,000. The Wassermann reaction has been found negative when reported.

The spinal fluid findings in epidemic encephalitis have led us to certain general conclusions in regard to the nature of the disease. The course of the disease, as illustrated by the serology of the spinal fluid, is extremely variable. A mild insidious or chronic type lasting for months, exists, as is shown by the continuation of abnormal changes in the spinal fluid over long periods of time. An acute fulminating type also exists and may or may not show serologic changes in the spinal fluid. Midway between these two extreme types of the disease is a combination of the acute, fulminating and chronic insidious types. In this type the spinal fluid findings indicate an active process which, after several weeks, subsides, leaving few or no serologic changes. Then the process lights up again with recrudescence of abnormal fluid findings, and may then continue to a fatal ending or what appears to be a recovery. It is striking that patients who have died have not, as a rule, shown marked aggravation of the spinal fluid changes before death. The reason is that death occurs from an involvement of vital centers, which is not dependent upon a meningitis.

The low average of cell counts found in this disease indicates that the meninges are very little involved in the pathologic processes, the point of attack of the infection being predominatingly through the vascular and lymphatic systems. The meninges are not directly attacked.

The diagnostic value of the spinal fluid findings in epidemic encephalitis is mentioned in detail in the section on differential diagnosis. The combination of an increase of cells, globulin and sugar with changes in the colloidal gold curve constitutes a tetrad of laboratory findings of diagnostic importance. Findings other than these are not of diagnostic importance unless related to the clinical findings.

The blood picture gives evidence of a type of infection which does not call forth a polymorphonucleosis. It does not differ essentially from the picture shown by other similar toxemias and is mostly of importance in differentiating from organic diseases of nontoxic origin, such as brain tumor. [Authors' Abstract.]

Marie and Bouttier. TREATMENT OF MYOCLONIC ENCEPHALITIS BY CICUTINE. [Bull. et Mém. Soc. Méd. des Hôp. de Paris, March 3, 1921. B. M. J.]

The symptoms observed after intoxication by cicutine (conine hydrobromide) are in every way comparable with, though much less rapid than, those caused by curare. Cicutine has a paralyzing action as long as the arterial circulation of the limb is preserved, but it has no action if this circulation has been interrupted by a high ligation. Marie and Bouttier have found that subcutaneous injection of cicutine in non-toxic doses has a sedative action on certain convulsive or spasmodic manifestations, especially those connected with lethargic encephalitis. They employ the following two different methods according to the cases: (1) If the symptoms are very marked, they start with a small dose and give only $\frac{1}{2}$ mg. the first day to test the susceptibility of the patient. A daily dose is then given for four or five days and increased from 1 to 3 mg. according to the state of the patient and the effect obtained. After five days the drug is usually suspended for two or three days, during which an examination is made of the urine, the reflexes, and the blood pressure. (2) When the myoclonic symptoms become less intense, but are still persistent and refractory to treatment, the injections are given only two or three times a week. The subsequent procedure depends upon whether there is a tendency to improvement, in which case the same doses are continued, or the reverse, when the dose is progressively increased if the drug is well tolerated. The writers have no doubt that there is a relation between the treatment employed and the results obtained, interruption of the injections being frequently followed by a reappearance or increase in amplitude of the myoclonic movements. On the other hand, cicutine has no effect on the choreiform movements of lethargic encephalitis or in ordinary chorea.

Langer, J. PARKINSONIAN SYMPTOMS AFTER EPIDEMIC ENCEPHALITIS. [Jahrbuch für Kinderheilkunde, 1921, XCVI, No. 1-2.]

This clinical study records three cases occurring in children. Hemorrhages, destruction of nervous tissue and calcification are evidently co-operating factors in the clinical pictures observed. The Parkinsonism developed a few weeks after the encephalitis, and rapidly reached its height, and has persisted unmodified during the more than a year up to the time of reporting the cases.

Hohman, L. B. PSYCHOSES OF EPIDEMIC ENCEPHALITIS. [Am. Archives of Neurology and Psychiatry, September 1921, VI, No. 3. J. A. M. A.]

Hohman analyzes the psychotic features of epidemic encephalitis as they have been observed in a group of twenty-three cases. A study of the psychotic data has made diagnosis possible in some patients who presented few neurologic signs, and the mental picture has been found

to offer valuable confirmatory evidence in doubtful cases with neurologic findings. Several characteristic symptoms have been made out as well as certain general reaction types. While the reaction types are not distinctive and decisive, they are often suggestive and helpful. Nine syndromes are presented in the order of their diagnostic importance.

Gelma, E. SIALORRHEA IN EPIDEMIC ENCEPHALITIS. [Paris Méd., March 19, 1921.]

This symptom, already commented on by others, is here discussed. It may be an early symptom and it may persist throughout the entire course of the disease. He finds it more frequent in children.

Scott, W. CASE OF ACUTE MYOCLONIC ENCEPHALITIS PRESENTING SOME UNUSUAL FEATURES. [Glasgow Med. Journal, February 1921.]

The outstanding features of the case cited by Scott were: The temperature remained normal throughout the illness, with the exception of a rise to 102.2 F. following lumbar puncture. The pulse averaged 120, the respirations 22 per minute. There were continuous clonic movements of the abdominal muscles on both sides, and early loss of abdominal reflexes. The spasmodic contractions were most marked on the right side of the body, and the main lesion was localized in the lower portion of the left precentral gyrus. There was continuous bleeding from the nasal and buccal mucous membranes; marked constipation and retention of urine. The patient died. At the post mortem examination a band of meningo-encephalitis was noted, extending over the left Rolandic area, and a similar condition was present in the corresponding area on the right side of the brain. The inflammation was especially marked over the left portion of the precentral gyrus on both sides, that on the left being more intense. [Author's abstract.]

Committee Report. [Bulletin de l'Académie de Méd., Paris, March 1, 1921. J. A. M. A.]

This committee report fills twenty pages and traces the history of epidemic encephalitis. It seemed to develop simultaneously in France and in Austria in 1916, although the war had interrupted all communication between them. It appeared in Australia also at the same time. It also prevailed in epidemic form in 1890 and in 1713, and probably existed in the Middle Ages and in antiquity. It is contagious but not violently so. The resistance of the virus is demonstrated by the long duration of the disease, the frequency of relapses, and the evidence that certain persons may transmit the disease several months or years after its first onset, and that healthy persons may be carriers. The secretions of the mouth and nose seem to be the vehicle of contagion, which suggests the possibility of infection by clothing, etc. Isolation should be maintained during convalescence but it is impossible to fix any date for the length of the isolation. Owing to the rarity of contagion, it is not necessary to be too anxious. Disinfection of the room, the

linen, and clothing is advised, and compulsory declaration of the disease and even of the dubious cases.

Cardenal H. G. de. SYDENHAM AND EPIDEMIC ENCEPHALITIS. [Jl. de Méd. de Bordeaux, February 25, 1921. J. A. M. A.]

Cardenal quotes from Sydenham's "Practice of Medicine" an account of an epidemic fever, 1673 to 1675, which seems to be identical with the present epidemic encephalitis. He kept the patients from lying down, fearing the congestion of the brain therefrom, and he described persisting hiccough as forming part of the clinical picture. He refers to mild nervous sequelæ of the disease as continuing for some time, but disappearing as strength was regained.

Wechsler, I. S. STATISTICS OF EPIDEMIC ENCEPHALITIS. [Neurol. Bull., Vol. III, p. 87, March 1921.]

This study was based on the records of 864 cases of epidemic encephalitis. Although most of them came from New York City practically every part of the country and Canada was represented, and included material from private practice and hospitals. The age incidence ranged from infancy to old age, the youngest being four, six and seven weeks and the oldest eighty-four. Childhood and adolescence were more or less spared compared to poliomyelitis, the period of greatest incidence being twenty to fifty. The male sex was more affected than the female, in the ratio of three to two. Occupation seemed to have no bearing on the disease. People of every walk of life seemed to have been affected. One curious fact was that seventeen physicians, almost two per cent of the group, were affected, a percentage entirely out of proportion to the numerical relation of the physician to the population. More than half of the patients (54 per cent) were foreign born. The majority of patients were married, which is explained by the age incidence of the disease. In twenty-two cases pregnancy complicated the disease. There were four fatalities in the group, a considerably smaller proportion than the deaths in influenza with pregnancy. Only in five instances of all cases reported did the disease occur in two members of one family. There was practically no contagion in hospital cases, although two interns had the disease. Direct contagion may therefore be considered a negligible factor. The mortality rate was less than 21 per cent, that is 178 out of 850 patients. The prognosis was generally more grave in the bulbar, psychotic, delirious, meningitic group, in patients with high fever and those with very acute onset. Death usually occurred early in the disease. While a great proportion recovered more or less completely, many patients showed sequelæ of the disease. There were many with residual paralysis agitans symptom; a great number of patients were left with ocular disturbances, tics, choreiform, myoclonic, or other abnormal involuntary movements. Epilepsy and psychic or even mental disturbances were occasional sequels. Twenty-six clinical

types of the disease are mentioned in the study. This profusion of classification reflects the diagnostic opinions of numerous observers. The lethargic, ophthalmoplegic formed the largest group, 35 per cent, and the Parkinsonian the next largest, 15 per cent. Many of the types or clinical forms could be put in six or eight fairly well-defined groups. [Author's abstract.]

Radovici, A. ACTION OF ATROPIN IN EPIDEMIC ENCEPHALITIS. [Presse Méd., January 29, 1921.]

The attenuation of the involuntary movements is shown in five cases of severe epidemic encephalitis under the influence of atropin. The doses were 1 or 2 mg, and all the patients experienced more or less relief from the choreiform jerking.

Levaditi and Harvier. EXPERIMENTAL ENCEPHALITIS LETHARGICA. [C. R. Soc. Biologie, February 12, 1921. J. A. M. A.]

These authors report that it possible to transmit the virus (which is preserved by passage through rabbits) to mice not only by intracerebral injection but, strangely enough, by intraperitoneal and subcutaneous injection, to which rabbits and other animals are refractory. In the case of mice there is an incubation period of two to three days after intracerebral injection and a period of eight days when the injection is made into the peritoneum or under the skin. The authors also record the production of encephalitic keratitis and transmission of the disease by corneal inoculation. An important finding is that the virus remains alive at room temperature for at least sixty days in milk and fifteen days in water. This renders plausible the supposition that water, and especially milk, may play the rôle of vectors of the virus in the propagation of epidemic encephalitis.

Levison, Ph. NEURO-ENCEPHALITIS (PARKINSONIAN TYPE.) [Ugesk. f. Laeg, February 24, 1921, Vol. LXXXIII.]

The author describes two cases of encephalitis with Parkinsonian symptoms.

1. One is a typical case of encephalitis lethargica in a boy of sixteen, where Parkinsonian symptoms appeared: a mask-like expression, rigidity and tremor. There appeared mors and microscopy showing extensive inflammation in the basal ganglions.

2. The second case was a child of nine, where suddenly appeared headache, vomition and severe perspiration; later on psychical disturbances and sleeplessness, and later still Parkinsonian symptoms: typically Parkinsonian carriage, walking (propulsion), hand position and tremor.

The author further mentions two other patients, who certainly had had influenza, but not acute encephalitis, and where there together with their illness appeared distinctly Parkinsonian symptoms as well as peripheral neuritis.

3. Woman of thirty-three. In February, 1920, had influenza. At first when she was going to get about again she had difficulty in walking, and in September, 1920, she appeared an almost typical picture of Parkinson's illness: expression of face, carriage, walking, rigidity and tremor (especially in the legs). The illness was complicated with polyneuritic symptoms in arms and legs (sensitive disturbances, swollen reflexes of tendo achilles, soreness of muscles and nerve branches).

4. Man of forty-four. In August, 1918, influenza, (mild case). About 1 month later, difficulty in walking and other movements. In February, 1920, Parkinsonian symptoms in expression of face, walking and carriage together with bad tremor of arms and legs, especially, however, action and intention tremor. The case was complicated with polyneuritic symptoms, as there were mild pareses, sensitive disturbances, swollen sinew reflexes as well as severe soreness of nerve branches and muscles. The patient has afterwards recovered.

The author especially draws the attention to the cases 3 and 4, which he has called cases of neuro-encephalitis (Parkinson's type) and thinks that the same noxe at the same time has attacked the central and the peripheral nerve system. That these have not been cases of genuine Parkinson is, among other things, shown by the age of the patients and the prognosis of the illness. The treatment has been diaphoresis, strychnin massage and motorpathy. [Author's abstract.]

Molnart. LETHARGIC ENCEPHALITIS. [Le Scalpel, April 30, 1921.]

The frequency of relapses often affecting quite a different part of the nervous system, in lethargic encephalitis is here commented on. The interval may be a considerable one. Hiccough or attacks of trigeminal neuralgia should not be grouped with encephalitis unless there is clear evidence of a primary attack of what is clearly encephalitis. Compared with poliomyelitis anterior, the lesions of encephalitis are much less degenerative in type, and consequently the prognosis is better. From their resemblance to those of trypanosomiasis and Sydenham's chorea, the author was led to try atoxyl in daily injections of 10 gr., and in the two cases reported he found it clearly beneficial. Encephalitis may have a peripheral localization, affecting the posterior nerve roots. In these cases there often persists some slight choreic movements, marked vasomotor and trophic troubles, and early swelling of joints. In this class of case sulpharsenol seemed of benefit.

Jones, B. L., and Raphael, T. THE PSYCHIATRIC FEATURES OF SO-CALLED LETHARGIC ENCEPHALITIS. [Am. Arch. Neurol. and Psy., February 1921, V, 150.]

Attention is directed to the fact that epidemic encephalitis has received relatively scant attention in the literature from the psychiatric aspect. Analyzed from this point of view, there appear to be two clinical stages in the evolution of the disease picture. The first, or irri-

tative stage, in which the mental picture is not unlike that found in other toxico-infectious conditions, is marked by a certain symptom heterogeny which may include any of the following features: euphoria, hypomania, depression, hallucinosis, paranoid reaction, or even states very simulative of the Korsakow complex. The second phase, responsible for the original designation, lethargic encephalitis, by Economo, is one essentially of psychosomnolence or apathy for which the authors suggest the term substupor. This phase is characterized by marked psychic torpor from which the patient, as a rule, may be aroused and which is often associated with pronounced catatonic features. In short, the keynote to the mental picture in this condition is attained in the conception of an essentially variable state dependent upon a toxico-infectious process, chiefly effective upon the basal ganglia, marked by an initial irritation period and dominated, at some time in the course of the disease, by a state of apathy or substupor in which the personality is fairly well preserved although definitely blurred by torpor. [Author's abstract.]

Thalhimer, William. EXPERIMENTAL AND CULTURAL STUDIES OF EPIDEMIC (LETHARGIC) ENCEPHALITIS. [Am. Archives of Neurology and Psychiatry, February 1921, Vol. V, pp. 113-120.]

Loewe and Strauss, with methods similar to those used by Flexner and Noguchi, in their investigation of poliomyelitis, demonstrated a filtrable virus of the central nervous system, nasopharyngeal washings and mucous membrane, spinal fluid, blood and autopsy material of cases of epidemic (lethargic) encephalitis. They injected this virus intracranially into rabbits and monkeys and produced the disease in these animals with typical pathological lesions. Many of these animals also showed a clinical picture practically identical with that of the disease. They cultivated from the above material and from central nervous system and nasopharyngeal mucous membrane of animals which died of the disease, a minute filtrable microorganism, similar in its cultural, morphological and staining characteristics to the so-called globoid bodies recovered by Flexner and Noguchi, from cases of poliomyelitis. They state that this organism differs in "virulence, occurrence, and particularly in its ability to infect rabbits" from the "globoid bodies" of poliomyelitis. The organism when injected intracranially into rabbits and monkeys also produced the disease and characteristic, pathological lesions in these animals. The same organism was recovered by these workers from the rabbits and monkeys which succumbed.

Levaditi and Harvier reported subsequently to the above mentioned communication that they also have caused monkeys, rabbits and guinea pigs to come down with the disease and obtained characteristic, pathological lesions after inoculation of the filtered virus from material of epidemic (lethargic) encephalitis cases. McIntosh and Turnbull, also secured positive results with a filtered virus injected into monkeys.

The epidemic (lethargic) encephalitis material on which the present investigation and report is based consists of two spinal fluids and four autopsies. Mandler filters, tested to hold back bacillus prodigiosus, were used in preparing filtrates from central nervous system. Rabbits were injected intracranially by the method of Loewe. Guinea pigs were also injected by a similar method. Cultures were made on ascitic fluid tissue medium, as perfected by Noguchi. The brains of the rabbits and guinea pigs which succumbed to the infection were removed with aseptic precautions. About 50 per cent of the rabbits succumbed after inoculation. They died usually in from ten days to three or four weeks. Some of them apparently died rather suddenly and were found dead in their cages, after having been apparently well the day before. Various types of palsy appeared in some of the rabbits, difficulty in swallowing, inability to swallow, typical myoclonus, etc. Palsy and apparent illness did not appear in guinea pigs, the animals being found dead in their cages after having been apparently well.

The central nervous system of the rabbit and guinea pigs showed typical lesions of epidemic (lethargic) encephalitis. These were not always the same and there was not always present the well marked perivascular round cell infiltration. In some animals only minute hemorrhages were present. In the cultures of ascitic fluid tissue media a minute, filtrable organism, identical in all respects with that described by Loewe and Strauss, was cultivated from the original material and from material secured from the animals. Cultures of this organism were injected into rabbits and also into guinea pigs and produced the disease with typical lesions. These animals did not die as quickly as those inoculated with the virus, as they usually lived from eight to twelve weeks after inoculation. The organism was again recovered from these animals. This organism has been carried through many generations. Some of the strains are present in the 12th generation. Control cultural studies were made of the medium used and of all the types of the material cultured, with negative results. Control animals were inoculated with materials similar to the various types used in the experiments. These animals continued well. Some of them were sacrificed and showed no lesions in their central nervous system.

Summary: The results of this investigation are confirmatory of both the experimental and the cultural studies of Loewe and Strauss. The studies of Loewe and Strauss, taken together with those of Levaditi and Harvier, McIntosh and Turnbull, and the results reported here, indicate that the filtrable organism cultivated and described by Loewe and Strauss, and secured from material from cases of epidemic (lethargic) encephalitis, is the etiological agent of this disease. [Author's abstract.]

BOOK REVIEWS

Freud, Sigmund. GROUP PSYCHOLOGY AND THE ANALYSIS OF THE EGO.¹ Translation by James Strachey. The International Psycho-analytical Library, No. 6. [The International Psycho-Analytical Press, London and Vienna, 1922.]

It is Freud's thesis that the bond which holds together the members of the herd is of the same general nature as that which holds together lovers, on the one hand, and hypnotist and subject on the other hand. This bond is love, variously manifested and disguised and more technically regarded as the emotional energy of the reproductive instincts. In all three of the above examples the sensual aspects of the love instinct, *i.e.*, the consciousness of their sexual origin and aim, are inhibited, *i.e.*, repressed or suppressed.

In the case of romantic infatuation, as everyone recognizes, they are sometimes only tentatively suppressed, at least in part; but in its tender and affectionate aspects, romantic love has been purged of conscious sexuality by repression. Similarly the hypnotized subject is held by this bond of transference, *i.e.*, sublimated libido, or love, entirely unconscious of its instinctive basis. In the case of the herd, Freud thinks a similar situation obtains, the members of the group being bound by love ties to a leader.

In nontechnical language this simply means that Freud regards the link binding the individuals in these various human relationships as a sublimated form of the same energy which is physically manifested as frank sexuality. So much of his thesis is new only in that it is applied to members of the herd as well as other human interrelationships. Freud does go on to make some new points, or rather to elaborate some previously made by him and other psychoanalysts.

The distinction is drawn between the love that would *have*, and the love that would *be*, *i.e.*, the love which reaches out for its object and claims it and possesses it, and the love which allows itself to be drawn out and become a part of the beloved, by emulation, imitation, etc. The former is known technically as object cathexis; the latter as identification. Identification is the original or earliest form of emotional tie with an object; subsequently it gives way increasingly to the predaceous, self-maintaining object-grasping type of love.

Regression to identification as a love linkage may occur, and does occur under both normal and pathological conditions. This Freud illustrates with a discussion of the origin of homosexuality, which in the male, for example, may arise from an identification of the patient with his mother, in which he psychically actually becomes she,

¹ Massenpsychologie und Ich-Analyse. Vienna, 1921.

and is in the position of desiring a masculine love object instead of a feminine one as would have been the case had he developed psychically from identification to object-cathexis love. In contrast thereto is melancholia, in which the object has been claimed by the ego, *i.e.*, object love has prevailed and has been brought to and identified with the ego, but here a strife has arisen between this ego and the ideal which the ego has set up for itself, the so-called ego-ideal. The latter then punishes the former for its shortcomings, whence come the bitter self-accusation, reproach, regret, and even suicide of the melancholiac. The ego-ideal is practically synonymous with the censor, as it was formerly called, the present view being that the ego-ideal has various qualities and functions, one of the latter being that of censoring or repression.

Freud carefully cites and discusses numerous other authors' opinions on the basis of herd instinct.

MENNINGER (Topeka)

Collins, Joseph. THE DOCTOR LOOKS AT LITERATURE. PSYCHOLOGICAL STUDIES OF LIFE AND LETTERS. [George H. Doran Company, New York.]

The "Doctor" has selected a rich material in which to make a psychological study. He has looked at it, however, with an often uncertain gaze. It is a gaze that has caught many a flash of illumination, which he has strikingly set forth, and one that has sometimes centered itself in a steadier contemplation of facts. But the doctor shows himself an inadequate portrayer of literary values, an untrustworthy psychologist, and since the latter science is basic to psychiatry, he fails to furnish an authoritative psychiatric background. He does indeed point out many an important truth which is illustrated in the varied range of literature he has selected, truth of human nature, in which he often presents at least something of its deeper psychological significance. In some chapters, as, for example, in the one on Dostoevsky, he gives a thoughtfully considered presentation of the author and his work. Yet even here the psychology fails to be sufficiently penetrating. It permits still those formal terms of the older psychiatry which savor of the dogmatism still possessing this "Doctor."

The fault lies mostly in his almost frivolous disregard of the more penetrating researches to which the "newer psychology" is more and more committing itself. This is more than disregard. Due to the doctor's own timidity or whatever cause, he actually falsifies the tenets of psychology by his misrepresentations of them as he touches upon them to reject them. This is true most conspicuously in regard to psychoanalysis, where his information seems most meager and yet to which he delights to make frequent reference. He reveals a manner of flinging facts from him which warps his point of view. Is a timidity at fault or is there rather a studied superiority to something which he will not take the trouble to understand seriously? At any rate it gives a character of dilettanteism to his work, for which

his frequent assertion of his own psychiatric experience cannot compensate.

This shallow incompleteness is most evident in his style. There is evidence that he possesses ability in literary expression, as in the telling brevity of a direct mode of speech, but the possibilities of this far too often are lost in a maze of confused sentences, clause piling upon clause in the unclearness of the author's thought. The style is weakened also by the frequent use of colloquialisms that add nothing to the effectiveness, which might atone for this offense against the dignity of literary purity. The existence of a "higher life, spiritual life," to be kept in mind by the literary writer as by the technical psychologist, is a self-imposed gospel which Collins feels bound to preach against the degeneracy of psychoanalysis. It is something of which he has found no indications in psychoanalysis as far as his knowledge of the latter goes. He seems to have forgotten that its existence is proved in a clear and pure literary expression. For him as in every other activity of the human mind, an effort toward an ideal reveals the release and the endeavor of the forces of life toward their best realization. Psychoanalysis could have taught him this greater goal and it would have given him fuller insight into the striving after this even in some of the literature he most condemns.

Spaulding, Edith R. AN EXPERIMENTAL STUDY OF PSYCHOPATHIC DELINQUENT WOMEN. Bureau of Social Hygiene. [Rand McNally & Co., New York.]

In 1912 a special laboratory hospital was created for the intensive study of the psychopathic delinquents of the Reformatory at Bedford, New York. Dr. Spaulding, who had had extensive experience at the Massachusetts Reformatory at Shelborne, was made its director. Dr. C. B. Shorer was the psychiatrist. This volume of nearly 400 pages would present a report of some of the work done so that others interested in similar undertakings might profit by the experience here recorded.

Seven chapters are devoted to the mechanism of the hospital, and six following chapters deal with the mental, physical and social problems involved. Special attention is given to the financing end of this experiment since such a consideration is not without value if similar ventures are to be thought of. In the clinical part of the book, Part II, the records of selected cases are recorded.

Ideal conditions were thought to be possible due to the munificence of the philanthropic interests of the founder. The plans are recorded. Conduct deviation was the chief problem studied. Other attempts, such as those at Framingham, Mass., are stated, and most of the issues are concisely dealt with. The case history material is unusually full and descriptive.

On the whole an excellent external view of a great effort which in a sense was not entirely carried to a finish, although it made an excellent effort.

Freud, S. DAS ICH UND DAS ES. [Internationaler psychanalytischer Verlag, Wien.]

Groddeck of Baden-Baden apparently first made use of the concept of the "das Es" as a conception of an aspect of the unconscious. Freud would seem to accept the term and has here given greater precision to the general conception.

"Das Es" he would offer as a term by which one can envisage a deeper layer of the unconscious than the "das Ich," which latter is capable of perceptions and relationships, of reasoning and of attaining consciousness; whereas in the "das Es" are those "instinctive," less personal emotional capacities. The Œdipus complex by identification and idealization makes a still further part of the personality, the "lieber-Ich." Here ethical strivings are represented, the conscience, etc.

In further discussion of these "regional," or "tensional" aspects of the unconscious Freud takes up again the ambivalent "Bios" of the Greeks. This large instinctive push he has separated into the Sexual Instinct, Eros, and the Death wish; the former conserving and extending life, the latter ending it in self or destroying it in others. These two instinctive trends are traceable back into cellular activities and psychoanalytic study shows their great significance in opening up new vistas for the study of the neuroses, particularly in epilepsy, where the forces bound up in the "das Es" may be glimpsed through the psychoanalytic technique. Although but a small monograph of 77 pages, it is an extremely compact presentation of great originality.

Freud, S. SAMMLUNG KLEINER SCHRIFTEN ZUR NEUROSENLEHRE. [Funfte Folge. Internat. psa. Verlag. Leipzig, Wien, Zurich.]

The fifth volume of Freud's collected contributions has appeared after a pause of four years since the large volume from Heller & Cie appeared. It contains six contributions, five of which are readily obtainable, but the first one, "Aus der Geschichte einer infantilen Neurosen," is here available. It was printed as the last contribution XXXII of Vol. IV and fortunately here reprinted since Vol. IV of the collected papers appeared during the war and has been somewhat difficult to obtain.

Special attention is directed to this striking analysis of an infantile neurosis obtained from a patient in his young manhood and in the course of treatment for other complaints. It is a gem and should some day appear in English with the Bruchstück and the Kleine Hans as a complete exposition of the technic of psa. II and III. Zur Vorgeschichte der analytischentechnik (Internat. Zeit. 1920) and III. Wege der Psa. Therapie (Int. Zeit. f. Psa. 1919) deal first with a short criticism of some misstatements of Havelock Ellis and some interesting observations concerning his own unconscious debts to earlier writers, and second Freud's address to the V Psychoanalytic Congress in Budapest, September, 1918, he speaks of newer pathways in psychoanalytic therapy. He elaborates the principle of

abstinence during psychoanalysis, and brings up the important question of the active participation of the patient in overcoming a compulsion, a very important issue in the treatment of the compulsion states.

The fourth paper on Homosexuality in the Female (*Int. Zeit. f. Psch.* 1920) has since appeared in English translation in the *International Journal of Psycho-Analysis*. V. *Ein kind wird Geschlagen* (*Internat. Zt. f. Psch.* 1919) offers some very important conceptions concerning the genesis of pederastic perversions, and in VI *Das Unheimliche* (*Imago*, 1919) Freud deals with some important aspects of esthetics, especially that feeling which is called strange, haunted, uncanny. The Tales of Hoffmann offer excellent material for this situation and Freud takes advantage of them.

Duret, H. TRAUMATISMES CRANIO-CÉRÉBRAUX (ACCIDENTS PRIMITIFS, LEUR GRANDS SYNDROMES). TOME III. DEUXIEME. VOLUME: LA COMPRESSION CÉRÉBRALE (SUITE) L'INTOXICATION HÉMATIQUE, L'HYPERTENSION INTRA-CRANIENNE. Recueilli et publié par J. Voituriez et J. Delépine. [Felix Alcan, Paris.]

This final contribution to Duret's monumental work on Traumatic Injuries to the Head and Brain is issued posthumously by two colleagues of the Lille faculty. We have had occasion in commenting on the previous volumes—a review is almost impossible by reason of the encyclopedic character of the work—to say we know of no study of its kind with which to compare it. It stands out as the most complete and thorough exposition of the large subject of cranio-cerebral injuries. It will remain a classic for all time by reason of its fullness of descriptive detail and because of the careful exposition of principles of surgical cerebral physiology and pathology. Although the author did not live to see his life work appear in such splendid form, his scientific immortality may be said to be assured.

Roffenstein, Gaston. ZUR PSYCHOLOGIE UND PSYCHOPATHOLOGIE DER GEGENWARTSGESCHICHTE. ARBEITEN ZUR ANGEWANDTEN PSYCHIATRIE, Vol. IV. [Ernst Bircher, Bern.]

There is much illumination to be gained for psychology and psychopathology as well as for the understanding of present social conditions if the principles of investigation and interpretation of psychology are turned upon the sociological field. The present writer has made a wide survey bringing together interpretative statements from sociologists and from psychiatrists or other psychological thinkers. He fails somehow in driving sociological or psychological facts into the heart of the problems of society and bringing forth a genuinely illuminating interpretation. His study is too discursive. His representation of the existence of a problem of present day history is not vivid. One feels that this is due to his failure to enter it as a problem of pressing economic value bound with the very dynamic impulses whose expression and whose needs are life itself.

Failure to thus unify his study as such a dynamic thing out of the very impulses of life itself belongs to his lack of understanding of the place of individual psychology as offering explanation as well as possible remedy. He does not leave individual psychology out of account but he conceives it too much only as an analogy from which to study mass psychology. This deprives him of the occasion of using it as a means of entering into the very factors which create mass psychology only out of the individual psyche and which make the acuteness of present conditions as well as men's varying attitudes toward them. Thus would he have had a warmer and more sympathetic appreciation both of the conservative and the radical groups. His really sincere endeavor to find psychology pertinent to the history of to-day would them have been more richly rewarded.

Stransky, Erwin. PSYCHOPATHOLOGIE DER AUSNAHMEZUSTÄNDE UND PSYCHOPATHOLOGIE DES ALLTAGS. ARBEITEN ZUR ANGEWANDTEN PSYCHIATRIE, Vol. III. [Ernst Bicher, Bern.]

Stransky presents a very interesting study of exceptional states whether in recognized psychopathic conditions or in the ordinary life of individuals, so-called normal people. He discusses the fact of marked differences in conduct at different times in the same individual which may manifest all degrees of contrast between courses of action up to a seeming split in the personality. He denies, however, behind any of these an actual split in the psyche but defines the situation rather as a displacement of the energy upon another constellation leading to activity. He speaks of a lability within the personality which permits the shifting of affect when endogenous or exogenous causes act upon the psyche to cause this varying direction of the energy. He writes, therefore, from the point of view of a dynamic psychology which gives actual explanation to the phenomena observed too often only in the external form of the manifest result. Although he acknowledges his sympathy with the psychoanalytic point of view he cannot yet free himself from the idea that it philosophically presents conceptions which are not based in fact. His own fruitful point of view could be further enriched could he perceive that there are facts which are the material of psychoanalysis which would explain still further what he has left unexplained. This is the nature of the affect which thus seeks displacement and the content of the constellations bound with it in the unconscious which determine such shift of energy.

Stekel, Wilhelm. DER FETISCHISMUS. STÖRUNGEN DES TRIEB- UND AFFEKTLEBENS (DIE PARAPATHISCHEN ERKRANKUNGEN) VII. [Urban und Schwarzenberg, Berlin, Vienna, 1923.]

Stekel has set forth with his accustomed fullness of detail many facts disclosed in the analysis of fetishism. His voluminous case histories, supplemented as they are by similar though unanalyzed histories from literature, presents a vast amount of material for review. Ample discussion is interspersed. Much that is valuable therefore is brought forward in that often striking manner by which

Stekel presses his thought and his material upon practical consideration. As always his aim is to extend the knowledge of the psychosexual life as a ground for the correction of individual difficulties and for a more helpful attitude toward them on the part of society, particularly of the medical and the legal portions of society. Fetishism is revealed as a phenomenon which belongs to normal sexuality in a moderate degree. But it is shown also as a very prevalent form of erotic interest narrowed to a limited field rather than healthily extended in full appreciation of the adult object of the opposite sex. Stekel finds it almost entirely confined to the male sex, representing there a flight from the female. It has its roots in some exaggeration of experience and of the erotic interests fixed upon such experience which has taken place in childhood. It is shown to be a form of compulsion and leads often to compulsive actions such as kleptomania or public exhibitionism. It is found in association with religion and becomes, according to the conception of the writer, a religion in itself. The writer's fertile fondness for interpretation has led him into rather phantastic explanation of the religion created by the fetishist, the attempt of the latter to make his fetish a synthesis of God and Satan. Stekel does not add to the scientific value of his book by carrying such explanation too far nor does he contribute as much that is original as he believes to the knowledge of the unconscious in his explanation of fetishism. Much of what he has said could have been left just as well within the terminology and under the interpretation which other psychoanalysts have already given it. Stekel's attempts to reexplain are not always convincing proof that the phenomena presented should be transposed into newly created categories. As a mass of material, however, the book presents valuable new matter.

N. B.—All business communications should be made to Journal of Nervous and Mental Disease, 64 West 56th St., New York.

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ORIGINAL ARTICLES

SENSORY DISSOCIATION IN SPINAL CORD LESIONS WITH NOTES ON SENSORY-PSYCHIC INTEGRATION

By JOSEPH BYRNE, A.M., M.D., LL.D.

DEPARTMENT OF BIOLOGY, FORDHAM UNIVERSITY; ATTENDING NEUROLOGIST,
CENTRAL NEUROLOGICAL HOSPITAL; CONSULTING NEUROLOGIST, FORDHAM
DIVISION, BELLEVUE AND ALLIED HOSPITALS, NEW YORK CITY

In a recent contribution (1) the author demonstrated the occurrence, after peripheral nerve injury, of two main forms of sensory dissociation viz. critical and protopathic. The present contribution shows that in spinal cord lesions these two forms of dissociation also occur.

CRITICAL DISSOCIATION

CASE 1. SYRINGOMYELIA; SENSIBILITY COMPLETELY ABOLISHED FOR THE AFFECTIVE OR HURT ELEMENT BUT PRESERVED FOR THE CRITICAL ELEMENTS (TOUCH, POINTEDNESS, SPECIFIC SENSATION OF HEAT [WARMTH] COLD, ETC.).

H. B., female, twenty-three years. When eighteen months old fell from window fracturing skull on left side. Afterwards paresis of right arm and leg. Operation for gastric ulcer when about nineteen years old. At twenty-one complained of severe headaches for which a decompressive operation was done by Dr. Wm. P. Sharpe. This relieved the headaches but had no effect on the right sided paresis. Shortly after the operation the left arm and hand grew stiff and weak and became insensible to pain, "heat," etc. Chief complaint: stiffness of left arm and hand; "sometimes can feel with left hand sometimes cannot"; left leg beginning to grow stiff. Laboratory findings irrelevant.

Examination (abstracted).—1. *Mentality*. Alert and bright; excellent coöperation.

2. *Motor*. Walks fairly well; left leg stiff; paresis of arms and hands, more so on left side; proximal phalanges of fingers flexed, distal phalanges extended; fibrillation and atrophy of interosseous

muscles of left hand; moderate scoliosis of thoracic spine; dermographia; legs "go to sleep" at times; never has tingling or "pins and needles" in arms or hands; legs and feet become restless and nervous at times; bladder and rectum normal.

3. *Sensory*. A. *Spontaneous*. Band sensation about middle of trunk; gastrointestinal discomfort at times when suffering from "indigestion"; no other pain. B. *Elicited*. I. SUPERFICIAL CRITI-

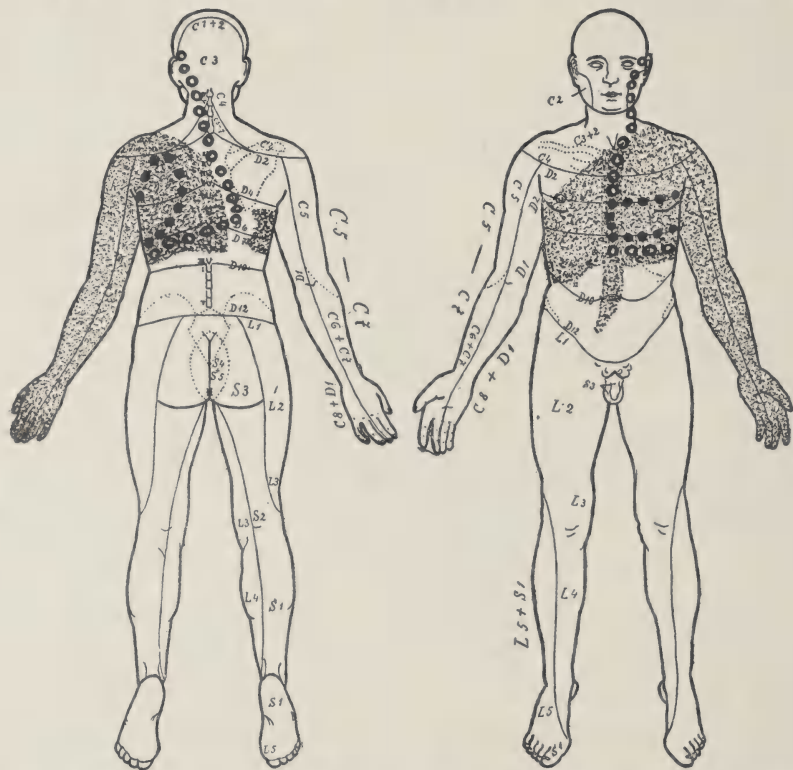


CHART 1. Case 1. Syringomyelia. Critical Dissociation. *Light dots*: loss for 0.5 gramme von Frey hair and warmth at 38° C. *Heavy dots*: bound loss for pressure-touch at 5.0 grammes. *Heavy circles*: bound loss for cold at 25° C.

CAL. Chart 1. (a) *Light touch*. Sensibility lost for von Frey hair * bending at 0.5 gramme pressure, on area of fine dots; preserved but impaired on unshaded areas; preserved everywhere for cotton wool (slightly dragged) sensibility after shaving (hands and forearms) being almost as good as before shaving; preserved everywhere for the heavier von Frey hairs but impaired on area bounded by large heavy dots (markedly) and on remainder of the skin (moderately) more so on right leg than left leg and more so on thighs than on legs and

* As the same hair was used in practically all the tests it was not considered necessary to represent the stimulus strengths in grm/mm², i.e., in energy units.

feet; localization impaired on right hand and arm, good on left. (b) *Warmth*. For 39° C. in silver tube with contact area of 3 sq. mm.: Lost on area closely coinciding with area of fine dots; impaired (introspectively) on unshaded areas, more so on right than left leg. (c) *Cold*. For 25° C. in silver tube: Lost on area bounded by circles; impaired on unshaded areas, more so on right than left leg. (d) *Compass points* (simultaneous). Impaired on hands and feet, more so on right especially on right hand.

II. SUPERFICIAL AFFECTIVE. Chart 2. (a) *Pricking*. For algesimeter at 3.0 grammes pressure: Absent for hurt element on shaded area preserved everywhere for critical elements (touch, pointedness, etc.); can distinguish point from head of a pin everywhere. Note area 8 by 1½ inches on top of head in which at times sensibility for the hurt element was preserved. A similar area was found in the vicinity of the right axilla where sensibility for the hurt element of pricking at 3.0 to 27.0 grammes was at times present. Area of hurt loss at 12.0 grammes a little smaller than that at 3.0 grammes. Over-reaction, present (trace) on face at boundary of hurt loss; present (trace) on trunk and lower limbs, more so on right.

Introspective evaluation for pricking at 4.0 grammes on hurt-loss area:

Touch.	++
Pointedness (Sharpness)	++
Hurt	0
Over-reaction	0
Radiation	0
Reference	0
Ability to name stimulus.	+

(b) *Heat*. For 45° to 70° C. in silver tube: Hurt element absent on area corresponding with area for prick loss at 3.0 grammes; area of loss for higher degrees slightly smaller than that for lower degrees; preserved everywhere for specific critical element (warmth); preserved on unshaded areas for hurt element. Over-reaction present (trace) on face and scalp at boundary of hurt loss and on trunk and lower limbs wherever the hurt element was preserved. Over-reaction greater on the right leg and foot than on left. Threshold for specific critical element (warmth) at 45° C. on left chest, including area bounded by heavy dots in Chart 1; on right chest within area of light dots in Chart 1 at 40° C.

Introspective evaluation for 62° C. momentary contact:

	Left Breast	Right Leg	Left Leg
Touch.	++	+ ♀ *	+
Warmth	++	+ ♀	+
Hurt	0	++	+
Over-reaction	0	+ (trace)	+ (trace)
Radiation	0	+ (trace)	+ (trace)
Reference	0	0	0
Ability to name stimulus.	+	+ ♀	+ ♀

* ♀ signifies impairment; ♀ ♀, marked impairment.

(c) *Cold*. For 20° to 0° C. in silver tube: Lost for hurt or unpleasant element on area corresponding with area of prick loss at 3.0 grammes; absent for specific critical element (cold) on left chest upon area bounded by heavy dots in Chart 1; preserved elsewhere for specific critical element; preserved for hurt element on unshaded areas. Over-reaction present (trace) wherever hurt element was preserved; more marked on right than on left leg and foot.

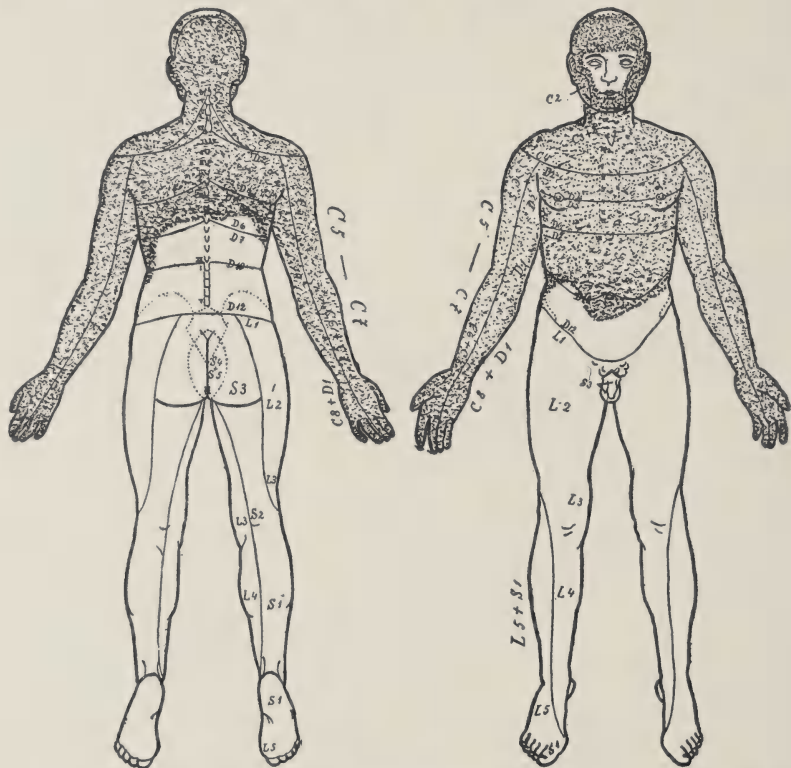


CHART 2. Case 1. Syringomyelia. Critical Dissociation. Shaded area: loss for hurt element of superficial and deep affective stimuli, e.g., pricking at 3.0 to 12.0 grammes, 70° C., etc.

Introspective evaluation for 0° C.:

	Area bounded by large dots Chart 1	Outside of area of large dots Chart 1
Touch.	++	++
Cold	0	++
Hurt.	0	0
Over-reaction	0	+ (trace lower limbs)
Radiation	0	0
Reference	0	0
Ability to name stimulus.	0	+

III. DEEP CRITICAL. (a) *Pressure-touch*. For aesthesiometer at 2.3 to 5.0 grammes pressure: Absent on area bounded by heavy dots in Chart 1; present elsewhere for 2.3 grammes; localization fair. (b) *Compass points* (consecutive). Impaired on hands and feet; more so on right. (c) *Posture and passive movement*. Impaired slightly. Appreciates movement in right hand and arm; well preserved in left hand and arm and at hips, knees, ankles and toes. (d) *Size, shape, weight and form*. Impaired in right hand; good in left hand.

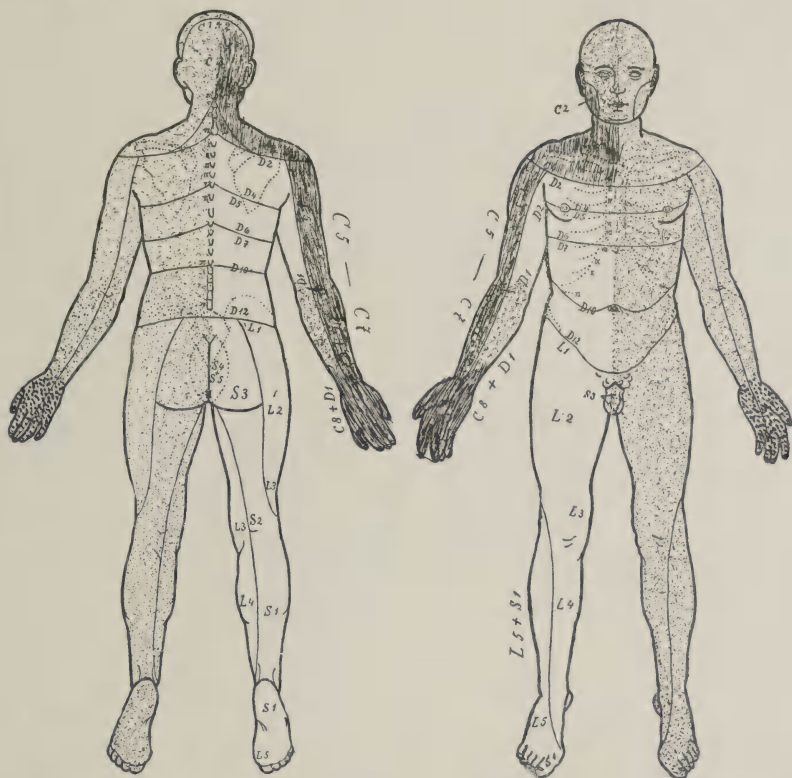


CHART 3. Case 2. Syringomyelia. Critical Dissociation. *Fine dots*: loss for 0.5 gramme and impairment for 2.0 grammes von Frey hair. *Heavy dots*: loss for 0.525 gramme von Frey hair. *Linear shading*: loss for 2.0 grammes von Frey hair and marked impairment for cotton.

IV. DEEP AFFECTIVE. Chart 2. (a) *Pressure-pain*. Lost for hurt element of all degrees of pinching, pressure, etc., on area closely coinciding with area of fine dots; preserved everywhere for critical elements and could name stimulus readily; preserved for hurt element on unshaded areas; over-reaction wherever hurt element was preserved, more marked on right than left lower limbs. (b) *Heat*. For 45° to 70° C. in large jug (slightly prolonged application). Hurt element lost on area closely corresponding with area of fine dots;

preserved everywhere for specific critical element (warmth); preserved on unshaded areas for hurt element with over-reaction (trace) more so on right. (c) *Cold*. For all degrees down to 0° C. in large jug, hurt or unpleasant element lost on area closely corresponding with shaded area; lost for specific critical element (cold) on area bounded by large dots in Chart 1; preserved for specific critical element elsewhere. Preserved on unshaded areas for hurt element with over-reaction, more marked on right than on left leg and foot. (d) *Vibration*. Absent on trunk on area of fine dots (Chart 1);

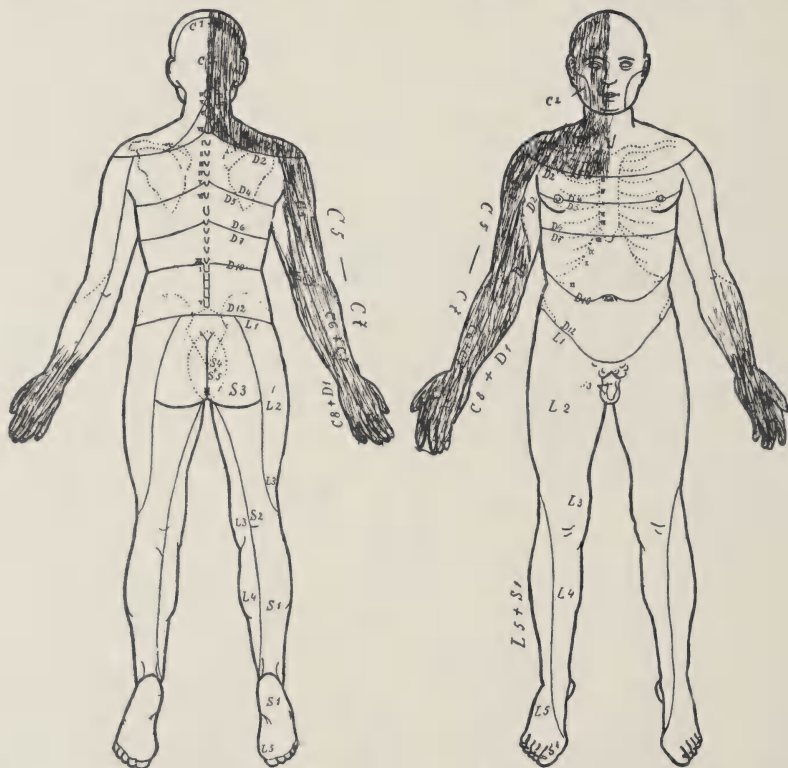


CHART 4. Case 2. Syringomyelia. Critical Dissociation. *Linear shading*: loss for hurt element in superficial and deep affective stimuli, e.g., pricking at 3.0 to 27.0 grammes, 70° C., ice, pinching, etc.

present on face and at hair margin on neck; absent on left limbs; present but impaired on right limbs. (e) *Hair sensibility*. For cotton lightly dragged over the hairy portions of chest, arm, face and hands: Lost for affective (tickle or unpleasant) element; preserved for critical element (moving contact). After shaving, sensibility still preserved for moving contact though to a less degree than before shaving.

4. *Reflexes*. As in spastic paralysis, e.g., ankle clonus, reversed plantar, etc., more marked in left than in right leg.

CASE II. SYRINGOMYELIA; DISSOCIATION SIMILAR TO THAT IN CASE I.

B. E., thirty-three years. Onset slow at thirty. Began on right side of head as "pins and needles" and passed to right shoulder, arm, and hand. Laboratory findings irrelevant.

Examination (abstracted). 1. *Mentality*. Good but tires easily.

2. *Motor*. Weakness of right arm and leg; seventh cranial normal; atrophy and fibrillary twitchings of right arm and hand; left upper arm becoming weak; right side of face and chest drier than left; ptosis of right eyelid; right pupil smaller than left (4.0:5.5 mm.).

3. *Sensory*. A. *Spontaneous*. Occasional aching pains in left arm and hand. B. *Elicited*. I. *SUPERFICIAL CRITICAL*. Chart 3. (a) *Light touch*. For cotton, impaired, linear shading; for 0.5 gramme von Frey hair, absent, linear shading and fine dots; for 0.52 gramme von Frey hair, absent, linear shading and coarse dots; for 2.0 grammes von Frey hair, absent, linear shading. *Localization*. Impaired, linear shading and coarse dots. (b) *Warmth*. Preserved everywhere for 39° C. in silver tube; impaired (trace) on shaded areas as compared with unshaded area. (c) *Cold*. Absent for 25° C., linear shading and coarse dots; trace of impairment fine dots. (d) *Compass points* (simultaneous). Impaired on cheeks and hands more so on right.

II. *SUPERFICIAL AFFECTIVE*. Chart 4. (a) *Pricking* at 3.0 to 27.0 grammes. Absent for hurt element linear shading; preserved everywhere for critical elements (touch, pointedness, etc.); can tell point from head of pin everywhere; over-reaction, subjective and objective, on unshaded area more marked on left side.

Introspective evaluation for pricking at 4.0 grammes on face:

	Right side	Left side
Touch	+	+
Pointedness	+	+
Hurt	0	++
Over-reaction	0	0
Radiation	0	0
Reference	0	0
Ability to name stimulus	+	+

(b) *Heat*. For 45° to 70° C. in silver tube, absent hurt element, linear shading; present everywhere for specific critical element (warmth); over-reaction on unshaded area especially on left side; threshold for specific critical element on right side of face at 38° C.

Introspective evaluation for 55° C. on face:

	Right side	Left side
Touch	++	+
Warmth	++	+
Hurt	0	+++
Over-reaction	0	0
Radiation	0	0
Reference	0	0
Ability to name stimulus	+	+

(c) *Cold*. For 22° to 0° C. in silver tube: Absent for hurt or unpleasant element, area closely corresponding with linear shading; absent for specific critical element (cold) on area closely corresponding with linear shading area; present for specific critical element elsewhere; over-reaction, unshaded area more marked on left side.

Introspective evaluation for 0° C. on face:

	Right side	Left side
Touch	++	+
Cold	0	++
Hurt or unpleasantness	0	+++
Over-reaction	0	0
Radiation	0	0
Reference	0	0
Ability to name stimulus	0	+

III. DEEP CRITICAL. Chart 3. (a) *Pressure touch*. Aesthesiometer at 2.3 grammes: Preserved with trace of impairment (introspective) on right side, linear shading and coarse dots. (b) *Posture and passive movement*. Moderately impaired on right side for elbow, wrist, fingers, hip, knee, ankle, and toes. (c) *Size, shape, weight, roughness*. Moderate impairment in right hand as compared to left

IV. DEEP AFFECTIVE. Chart 4. (a) *Pressure-pain*. Absent for all degrees of pinching, pressure, etc., on area closely corresponding with linear shading; preserved everywhere for critical elements and could name stimulus; over-reaction on unshaded area more marked on left side. (b) *Heat and cold* in massive prolonged application. Sensibility absent (hurt element), on shaded area; over-reaction on unshaded area more marked on left side. (c) *Vibration*. Impaired for affective element on right side (face, arm and hand); vibration rate faster on left than right hand; impaired on legs and feet more so on right.

4. *Reflexes*. As in spastic paralysis, signs more marked on right side.

CASE III. EXTRADURAL CARCINOMA (METASTATIC) CAUSING PARAPLEGIA IN FLEXION WITH SUPPRESSION, BELOW WAIST, OF ALL FORMS EXCEPT DEEP AFFECTIVE WHICH WAS RETAINED WITH PROTOPATHIC CHARACTERISTICS; OPERATION; SPONTANEOUS PAINS WITH RESTORATION OF SENSORY FUNCTION; PERIODS OF REGRESSION CAUSED BY TOXIC ABSORPTION; SUPERFICIAL AND DEEP PROTOPATHIC DISSOCIATION.

H. C., female, forty-seven years, widow. Chief complaint: Numbness and loss of power from the waist down. Onset in December, 1916, with pains in thighs (roots Lii) which persisted with intermissions until May, 1917, when patient was confined to bed. About July 20, 1917, she lost the power in both legs. On July 24, 1917, she entered Fordham Hospital, service of Dr. John J. McGrath, suffering from strangulated ventral hernia which was effectually dealt with in an emergency operation by Dr. Anthony H. Harrigan. On admittance a small bed-sore was present over the sacrum. Labora-

tory reports: Urine, normal; x-ray of spine, negative; blood and spinal fluid, no examination made.

Examination August 10, 1917. 1. *Mentality.* Unusually alert and intelligent; excellent coöperation.

2. *Motor.* Paraplegia in flexion. Each limb draws up suddenly in bed, apparently spontaneously. Urinary retention necessitating periodic catheterization; bowel incontinence; spontaneous evacuation without warning to patient.

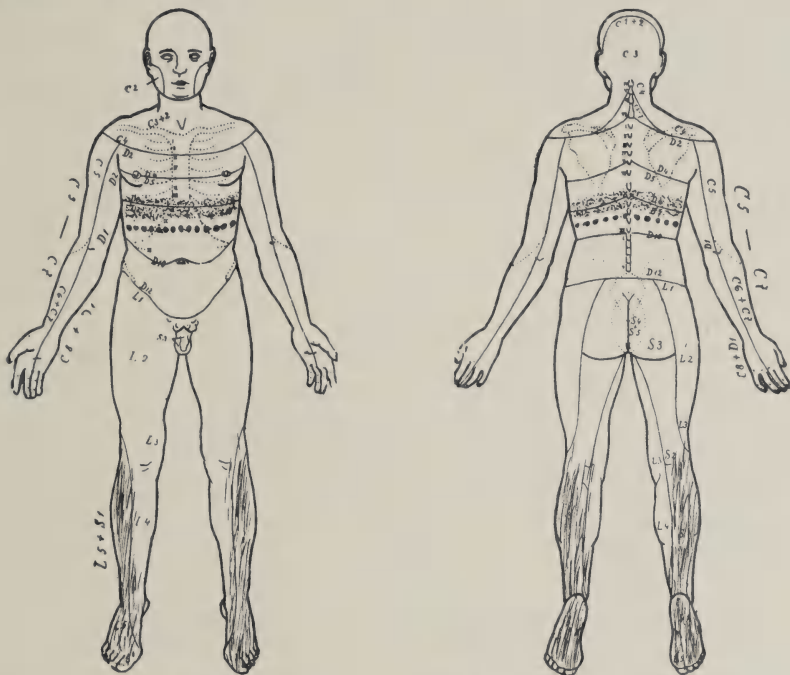


CHART 5. Case 3. Extradural neoplasm. Before operation. Protopathic dissociation from remote cord compression. *Light stippling*: impairment for 0.5 gramme von Frey hair. *Heavy dots*: bound loss for pricking at 3.0 grammes; hyperalgesia for $2\frac{1}{2}$ inches above line of dots. *Linear shading*: deep affective sensibility retained with protopathic characteristics.

3. *Sensory.* A. *Spontaneous.* Numbness from the waist down: slight pain "in the muscles of the center of the back," i.e., between, or just below, the angles of the scapulæ. B. *Elicited.* I. *SUPERFICIAL CRITICAL.* (a) *Light touch.* Sensibility was lost for von Frey hair bending at 0.5 gramme pressure, as set forth in Chart 5. (b) For *warmth* (39° C.) and *cold* (25° C.) in silver tubes, sensibility lost over area closely corresponding with that for von Frey hair loss.

II. *SUPERFICIAL AFFECTIVE.* (a) For *pricking*, with algesimeter at 3.0 grammes pressure, sensibility lost over area slightly less than that for the 0.5 gramme von Frey hair loss. Pricking at 27.2

grammes lost below level of root thoracic X. Upon soles and outer aspect of either leg this stimulus evoked flexion reflex (objective over-reaction). Over-reaction for dragged pin point present in zone two inches wide just above boundary of loss for pricking at 3.0 grammes. (b) *Heat*. For 62° C. in silver tube (light momentary contact) lost, generally speaking, up to level of loss for pricking at 3.0 grammes. On outer aspect of left leg preserved for hurt element of this stimulus; radiation and reference (to knee and sole) present. Over both soles, upon slightly prolonged application of stimulus, sensibility preserved for hurt element and to a much less degree for the specific critical element (warmth). The sensation was poorly localized besides being attended by spreading, subjective over-reaction (trace) and objective over-reaction (flexion reflex). Introspective evaluation for 62° C., applied to soles and outer aspect of left leg:

Touch	0
Warmth	+ (trace on sole)
Hurt	++
Radiation and reference.....	+
Subjective over-reaction	+
Objective over-reaction	+
Ability to name stimulus.....	0

(c) *Cold*. For ice and salt in silver tube sensibility lost over area corresponding to loss for 62° C. Sensibility for hurt (burning, "electric") element preserved on soles and outer side of left leg. On soles, subjective and objective over-reaction. Applied to either sole 78° C. hurt more than ice and salt, but where latter was applied immediately after removal of 78° C. the cold caused added discomfort and seemed to be, under these circumstances, the more effective stimulus.

III. DEEP CRITICAL. (a) *Pressure-touch*. For aesthesiometer, at 2.3 grammes pressure, lost below root thoracic VIII. (b) *Posture and passive movement*. Absent in both limbs at knees, ankles and toes. (c) *Size, weight, compass points and roughness*, lost.

IV. DEEP AFFECTIVE. (a) *Pressure-pain*. Over anterior tibial group, algometer threshold averages: Right leg 9.0 kilos; left leg 8.0 kilos. Subjective over-reaction, spreading, reference (to knee region), and inability to name stimulus. For *deep pinching* of skin on outer aspect of either leg, retained for hurt element; radiation, reference (to hypochondrium and hypogastrium), poor localization, objective over-reaction, subjective over-reaction (trace), inability to name stimulus. Forcible flexion of great toe evoked indefinable pain referred to knee. *Rapidly repeated pricking* at 30.00 grammes, retained, on soles and outer aspect of each leg, for hurt element; radiation, reference, objective over-reaction, subjective over-reaction (trace), impaired localization, inability to name stimulus. *Single pricks* on sole and outer leg at 30.00 grammes, evoked no unpleasant sensation but elicited the flexion reflex (objective over-reaction). (b) *Heat, in massive more or less prolonged application*. Water at 50° C. in large jug felt over legs and thighs as "something" with

trace of unpleasantness. At times patient felt it on outer calf as "hot." Over soles stimulus evoked stinging sensation poorly localized and spreading widely. No subjective over-reaction but stimulus on soles and legs evoked flexion reflex. Boundary of loss for critical element (warmth) of stimulus lay two inches below that for pricking at 3.0 grammes on left side and one inch below this boundary on right side. (c) *Cold*. For iced water in large jug lost for specific element (cold) at two inches below level of loss for pricking at 3.0 grammes; on soles, legs and thighs stimulus felt as "something" with trace of unpleasantness but patient could not name stimulus; poor localization, wide radiation; no subjective over-reaction but stimulus readily evoked flexion reflex. Evaluation for elements of ice on soles:

Touch	0
Cold	0
Hurt or unpleasantness	+
Radiation and reference	+
Subjective over-reaction	+
Objective over-reaction	+
Ability to name stimulus.....	0

(d) *Vibration*. Absent below costal arches.

4. *Reflexes*.

	Right	Left
Epigastric	0	+
Abdominal	Not tested	Not tested
Anal	+ ♀*	+
Elbow	+	+
Wrist	+	+
Knee (with reën- forcement)	0	0
Ankle	0	0
Ankle clonus	0	0
Plantar	H,O;R,♀; knee flexes (trace)	A,♂*; flexion reflex
Oppenheim	H,O;R,♀; knee flexes	A,♂; flexion reflex
Gordon	H,O;R,♀; knee flexes	A,♂; flexion reflex

Myotatic irritability preserved in both legs and thighs.

Summary. 1. *Motor*. Paraplegia in flexion; loss of sphincteric control.

2. *Sensory*. *Superficial* and *deep critical* suppressed to waist level. *Superficial affective* suppressed below waist except over soles and outer aspect of left leg. *Deep affective* retained (slightly impaired) in legs and feet for hurt element, with some protopathic characteristics.

3. *Reflexes*. As generally found in almost, but not quite, complete functional interruption in cord above lumbar segments, *i.e.*, partial reversion to flexion reflex.

*In testing the plantar reflex and other modifications of the flexion reflex, *e.g.*, the Gordon (deep calf pressure), Oppenheim (stroking along inner border of tibia), etc., ♀ signifies downward movement of toe or toes; ♂, upward movement; H, hallux; R, rest of toes; A, all the toes. For any other reflex, ♀ signifies impairment; ♀♀, marked impairment; ♂, exaggeration; and ♂♂, marked exaggeration.

Anatomical diagnosis. Lesion extending from seventh or eighth thoracic to first lumbar segment.

Etiological diagnosis. Extramedullary neoplasm (carcinoma). Operation by Dr. E. R. Cunniffe August 31, 1917. Laminectomy of eighth to the eleventh thoracic vertebrae. Beneath the softened and eroded laminae was found a moderately hard, nodulated mass which bulged outwards as the upper laminae were removed. Tumor removed by morcellation. It surrounded the cord for a distance of two or three segments but did not involve the dura mater otherwise than by directly compressing it and its contents. The dura was not opened. After operation the patient was examined at frequent intervals to determine the order of return of the sensory components.

Examination on *second day* after operation:

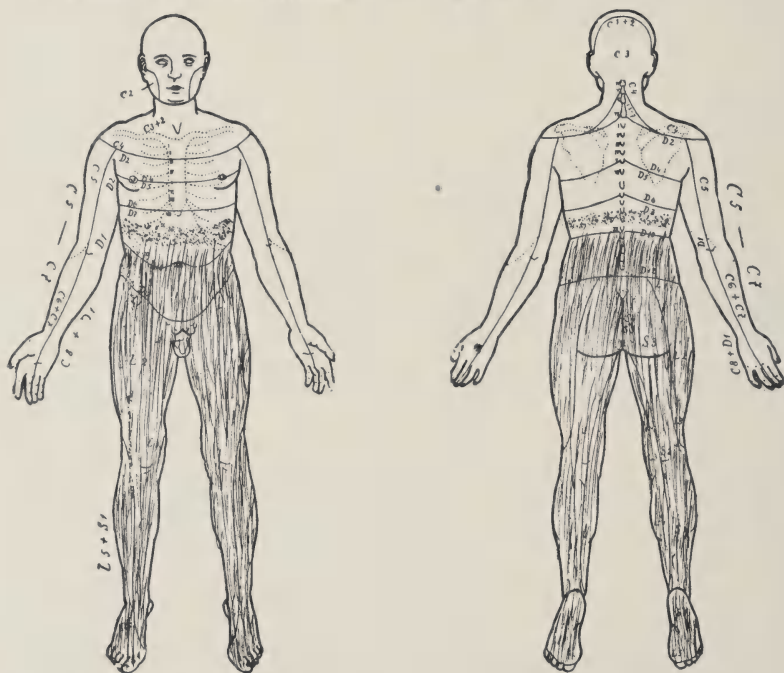


CHART 6. Case 3. On the fifth day after operation. Superficial affective sensibility restored. *Light stippling*: impairment for superficial critical stimuli separating normal sensibility above from lost sensibility below. *Linear shading*: restored sensibility, with protopathic characteristics, for superficial affective stimuli.

1. *Mentality.* Excellent.
2. *Motor.* Voluntary movement still absent in lower limbs; spontaneous movements (flexion reflex) somewhat diminished.
3. *Sensory.* A. *Spontaneous.* Numbness as before operation. B. *Elicited.* I. *SUPERFICIAL CRITICAL.* (a) *Light touch.* For the 0.5 gramme von Frey hair, sensibility absent on front of lower limbs up to groin (twelfth thoracic root) on both sides. Above this, sensi-

bility impaired up to level of eighth thoracic root. On posterior aspect of left thigh, just below gluteal fold, sensibility retained, with some impairment, over area about $1\frac{1}{2}$ inches in diameter. With this exception sensibility was absent posteriorly on both sides up to the level of the twelfth thoracic root and impaired from twelfth thoracic up to the level of eighth thoracic root. (b) Other superficial critical forms not tested.

II. SUPERFICIAL AFFECTIVE. (a) For *pricking*, at 27.2 grammes, sensibility absent to level of twelfth thoracic root on left, the level of loss being a little higher on right side. At boundary of preserved sensibility nature of stimulus was well appreciated. (b) *Tests for heat and cold* not made but sensibility was absent all over lower limbs as seen in the deep affective tests for heat and cold.

III. DEEP CRITICAL. *Pressure-touch*. (a) For aesthesiometer, at 2.3 grammes, sensibility absent on front and back of limbs to level of great trochanters with exception of area two inches in diameter on the posterior aspect of left thigh, just below the gluteal crease. (b) *Posture and passive movement*. Tests showed gross impairment for ankles and toes just as before operation.

IV. DEEP AFFECTIVE. (a) *Pressure-pain*. Pressure on calf muscles evoked pain much more readily than before operation. Typical protopathic characteristics, *e.g.*, sudden entry into consciousness, spreading, reference, over-reaction (subjective and objective), and absolute inability to name stimulus. No measurements made. (b) *Heat*. For 80° C. in large jug sensibility present for the hurt element on soles, legs and thighs, with protopathic characteristics, up to level of twelfth thoracic root. Over posterior part of left thigh upon area three inches in diameter, just below gluteal crease, the stimulus was felt as hot. Subjective over-reaction for this stimulus, as well as for pressure-pain, though more in evidence than before operation was not very marked. (c) *Cold*. For *ice* in large jug sensibility for specific element (cold) absent to level of twelfth thoracic root. For affective (hurt or disagreeable) element sensibility present on soles and thighs, and to a less degree on legs, with protopathic characteristics. (d) *Vibration*. Sensibility returned (quality of sensation) upon the tibia, more so on right than on left. Patient recognized similarity between sensation on legs and on hand but sensation felt in legs was of greater intensity and extent (spreading) and the vibrations of slower rate. Localization very poor. (e) *Roughness*. Sandpaper, and scraping of the soles, evoked sensation of "numbness" not unpleasant but poorly localized and associated with persistence, and reference (to the anterior tibial region). Cotton-wool dragged over the hair of legs and thighs evoked same sensation as sandpaper.

Summary. Noteworthy features on the second day: (1) Return of *superficial critical* and *affective* sensibility on lower portion trunk. (2) Lowered threshold on legs and thighs for hurt element of *deep affective* stimuli; sensibility unchanged, *i.e.*, absent, for critical elements of these stimuli, as well as for all elements of *superficial* and

deep critical stimuli, and of superficial affective stimuli. (3) Markedly lowered threshold for deep affective stimuli applied to soles.

Third day. Sensibility present in legs and soles, with marked protopathic characteristics, for deep affective stimuli, e.g., pressure-pain, 55° to 60° C., cold (ice), tuning fork, and scraping. Scratching, and rapid pricking of sole at 27.2 grammes, elicited the flexion reflex and a disagreeable sensation, the nature of stimulus not being appreciated. Sensibility absent on legs and feet for superficial critical and affective, and for deep critical stimuli.

Fourth day. Sensibility returned for some forms of deep critical stimulation. *Pressure-touch.* Average of thresholds were: Outer ventral aspect of right thigh, 6.75 grammes; ventral aspect of thighs and legs, 9.1 grammes; dorsum of left foot, 41.7 grammes. Sensibility for this stimulus (41.7 grammes) absent on right foot. *Compass points* horizontally, at separation of three inches, to ventral aspect of either thigh, both simultaneously and consecutively, were felt as two when applied with sufficient force to cause pain but not when the sensation of pressure-touch only was evoked. Deep affective sensibility was about as it had been on third day with exception that 65° to 70° C., in massive application, was just beginning to be felt as warm. Return of sensibility for specific (critical) element of this stimulus significant in light of returned sensibility for pressure-touch. Threshold becoming lower, day by day, for deep affective stimulation, e.g., painful pressure on calves, tuning fork. No appreciable improvement in posture and passive movement.

Fifth day. 1. *Mentality.* Good.

2. *Motor.* No return of function in limbs; has recovered some control over anal sphincter.

3. *Sensory.* A. *Spontaneous.* From day to day patient becomes more and more aware of existence and posture of her lower limbs. For past two days she knows when her bowels want to move and is aware that she can exert some voluntary control over the anal sphincter. B. *Elicited.* I. SUPERFICIAL CRITICAL. (a) *Light touch.* Sensibility for 0.5 gramme von Frey hair, absent up to level of ninth thoracic root. Chart 6. (b) *Warmth* (38° C.) and *cold* (25° C.), sensibility absent up to level of ninth thoracic root. (c) The *compass points*, at a separation of two inches, applied simultaneously and longitudinally above the knee were appreciated fairly well.

II. SUPERFICIAL AFFECTIVE. (a) For *pricking*, at 2.3 and 27.2 grammes, sensibility had returned for hurt element, with typical protopathic characteristics, over the thighs, legs, and feet. Nature of stimulus not appreciated up to a level just below lower boundary of loss for light touch. (b) *Heat.* For 70° C., lightly applied, sensibility returned, with protopathic characteristics, for hurt element all over the thighs, legs and feet. The stimulus in order to be felt required somewhat more prolonged application than that usually made in this type of test. Subjective over-reaction present but not marked. The specific element not appreciated up to level cor-

responding roughly with lower boundary of loss for light touch. Evaluation for elements of this stimulus (70° C.) applied to the legs and feet stood:

Touch	0
Heat (Warmth)	0
Hurt	++
Radiation and reference	+
Subjective over-reaction	+
Objective over-reaction	++
Ability to name stimulus	0

(c) For *cold* (ice and salt) sensibility returned for disagreeable element, with protopathic characteristics, over thighs, legs and feet. Specific element (cold) not consistently appreciated up to level corresponding with lower boundary of light touch loss. Over foot, leg and thigh the stimulus was most frequently felt as hot, being felt as very hot at level of eleventh and twelfth thoracic roots. Occasionally it was felt as cool on outer aspect of right calf. Marked over-reaction, subjective and objective, for superficial affective stimulation (pricking, 55° C., ice, and dragged pin-point) over lower abdomen (eleventh and twelfth thoracic roots).

III. DEEP CRITICAL. (a) *Pressure-touch*. Sensibility for 41.7 grammes present all over both lower limbs and feet. Localization good. Threshold values slightly lowered as compared with the results of yesterday. (b) *Posture and passive movement*. Sensibility returned to a considerable degree for joint movement, *e.g.*, in the knees and ankles, and to a less degree in the toes, although the improvement in the latter was quite noticeable. (c) *Size*. Recognized difference between leather disks measuring one and two inches respectively when these were successively pressed against the sole. (d) *Weight*. Appreciated increment and decrement when a weight of 100 grammes was added to, or taken from, a weight of 200 grammes resting on the dorsum of the fully supported foot.

IV. DEEP AFFECTIVE. (a) *Pressure-pain*. Localization good, the threshold averages being: Anterior tibial region of left leg, 2.5 kilos; calf of left leg, 4.0 kilos. Protopathic characteristics had largely disappeared especially the spreading. Subjective and objective over-reaction more controlled. This was in marked contrast to the condition of superficial affective sensibility in which protopathic characteristics had just appeared. (b) *Heat*. For water at 70° C., in large jug, sensibility returned over legs and especially over soles for the specific element (warmth) whereas the protopathic characteristics attending the hurt element had abated somewhat, the spreading being less and the localization better. Improvement in appreciation of nature of stimulus. (c) *Cold*. For *iced water*, in large jug, sensibility present, with protopathic characteristics, over thighs, legs and feet. The specific element not appreciated. (d) *Vibration*. Sensibility present and equal upon both legs with some slowing in the rate of vibration as compared with the hand. (e) *Scraping* (finger nail). Sensibility in legs and feet approaching normal.

(f) *Roughness* (sand paper). Sensibility approaching normal in legs, but somewhat impaired in feet.

4. *Reflexes.*

	Right	Left
Epigastric	0	0
Abdominal	0	0
Anal	++	++
Elbow	+	+
Wrist	+	+
Knee	0	0
Ankle	0	0
Ankle clonus	0	0
Plantar	H,O;R, ♀	H,O;R, ♀
Oppenheim	H,O;R, ♀	H,O;R, ♀
Gordon	H,O;R, ♀	H,O;R, ♀

Summary. 1. *Motor.* Further signs of returning anal control.

2. *Sensory.* A. *Spontaneous.* Awareness of position of limbs returning. B. *Elicited.* I. SUPERFICIAL CRITICAL. Absent on lower limbs. II. SUPERFICIAL AFFECTIVE. Returned on lower limbs with protopathic characteristics. III. DEEP CRITICAL. Almost completely restored. IV. DEEP AFFECTIVE. Has lost most of its protopathic characteristics.

3. *Reflexes.* Replacement of flexion reflex and upward movement of toes, upon plantar stimulation, by partial return to the normal flexor response, *e.g.*, H,O;R, ♀.

Sixth day. 1. *Mentality.* Good.

2. *Motor.* As on fifth day.

3. *Sensory.* A. *Spontaneous.* As on fifth day. B. *Elicited.* I. SUPERFICIAL CRITICAL. (a) *Light touch.* For the 0.5 gramme von Frey hair, sensibility absent up to level of tenth thoracic root. (b) For *warmth* (38° C.) and *cold* (27° C.), sensibility absent up to level of loss for light touch. (c) *Compasses.* Points separated 2¾ inches, and applied simultaneously and horizontally to front of lower third of left thigh, the answers in ten trials were: for "ones," 5 right, 3 wrong, and 4 no answer; for "twos," 5 right, and 5 wrong.

II. SUPERFICIAL AFFECTIVE. For *pricking*, at 27.2 grammes, in single pricks, sensibility present upon legs, thighs and feet for hurt element, with protopathic characteristics, *e.g.*, spreading and inability to name stimulus. No marked over-reaction. At level of eleventh thoracic root the nature of the stimulus was recognized and, for a short distance below this, over-reaction, subjective and objective, was marked for pricking and the dragged pin-point. (b) *Heat.* For 42° C. sensibility present on front of thighs for hurt element. Nature of stimulus not recognized up to two inches below boundary of loss for light touch. Water at 65° C. was felt as pricking and burning above the knees on momentary contact. No marked over-reaction for pricking or for 65° C. The other protopathic characteristics were present, *viz.*, spreading, etc., and inability to name stimulus. (c) For *cold* at 23° C. sensibility present, with protopathic characteristics for hurt element, above the knees. Two inches below boundary of light touch loss this stimulus was felt as "hot," and was

not felt as cold until the boundary of light touch loss had been reached. For 21° C. and for ice, the hurt element was felt, with protopathic characteristics, on soles and front of the thighs but not on legs. Nature of stimulus not appreciated up to level two inches below boundary of light touch loss.

III. DEEP CRITICAL. (a) *Pressure-touch*. For 2.3 grammes sensibility absent up to one inch below boundary of light touch loss. For 41.7 grammes, sensibility present above knee on both sides (front of thighs), and above ankle on left side. Localization good. (b) *Posture and passive movement*. Sensibility fairly good in knee and ankle but still considerably impaired in toes. After a few trials (reëducation), the answering for position and movements of toes was quite good. (c) *Weights*. Above knee patient readily appreciated increment and decrement when 50 grammes was added to, or taken from, 100 grammes. On dorsum of supported foot patient could not appreciate 100 grammes added to, or taken from, 200 grammes.

IV. DEEP AFFECTIVE. (a) *Pressure-pain*. Threshold average at 2.2 kilos over anterior tibial region of both legs and on calf of left leg. Localization good; gradual entry into consciousness; no over-reaction. Pinching leg, and deep pressure with the finger tips, caused sensation of hurt which was not attended by over-reaction. Nature of both stimuli well appreciated. (b) *Heat*. Water at 83.5° C. in large jug, felt as hurt over legs and soles; as sticking above knees; and as "hot" on the middle of thighs in front; but was not in any of these tests attended by marked over-reaction even on fairly long contact. (c) *Cold*. *Iced water* in jug felt as unpleasant over feet and legs. On upper part of thigh felt as "burning." No over-reaction upon soles and legs. (d) *Roughness*. Sensibility on first trials good above knees but defective in later trials (fatigue).

Summary. I. SUPERFICIAL CRITICAL. Absent on thighs, legs, and feet.

II. SUPERFICIAL AFFECTIVE. Returned to great extent on thighs for hurt element of heat and cold; and on thighs, legs, and feet for hurt element of pricking.

III. DEEP CRITICAL. About as on fifth day.

IV. DEEP AFFECTIVE. About as on fifth day.

Seventh day. Patient felt nervous and feared she would not do so well with tests. Sensibility found in legs and feet as follows:

I. SUPERFICIAL CRITICAL. Absent.

II. SUPERFICIAL AFFECTIVE. For *pricking* at 27.2 grammes, and for *heat* at 65° C. present with protopathic characteristics; for *pricking* at 3.0 grammes pressure, absent.

III. DEEP CRITICAL. In general not so good as on the fifth and sixth days although 80 grammes added to, or removed from, 100 grammes resting on the supported foot, was well appreciated.

IV. DEEP AFFECTIVE. For heat and cold the protopathic characteristics were somewhat more marked than on the fifth and sixth days.

Ninth day. Spontaneous, intermittent, shooting pains like those of tabetic crises were felt for the first time all over the thighs, legs and feet.

Tenth day. The shooting pains persisted, chiefly in feet and legs, and not so much in thighs.

1. *Mentality.* Good.

2. *Motor.* Cannot move feet, legs or toes; has some control over bladder and rectal sphincters.

3. *Sensory.* A. *Spontaneous.* Intermittent shooting pains in feet and legs but not so much in thighs; knows when she wants to pass urine or defecate; has a positive sensation of numbness in feet for first time to-day. B. *Elicited.* I. SUPERFICIAL CRITICAL. (a) *Light touch.* Sensibility for the 0.5 gramme von Frey hair absent on dorsum and sole of each foot, and on each leg up to level of fourth lumbar root. (b) *Warmth.* For 39° C. sensibility absent to level of light touch loss. (c) *Cold.* For 25° C. sensibility absent for specific element up to level of tenth thoracic root. This stimulus which, under the circumstances acted like a superficial affective stimulus, was felt as "hot" on lower abdomen and groin (eleventh and twelfth thoracic roots). (d) *Compass points* at separation of 3½ inches, applied simultaneously and longitudinally on the thighs and legs, not appreciated as two. The spontaneous pains interfered with the tests.

II. SUPERFICIAL AFFECTIVE. (a) For *single pricks* at 3.0 grammes sensibility absent up to level of boundary of light touch loss (fourth lumbar root). Some spreading and over-reaction, subjective and objective, still in evidence upon front of thighs, although nature of stimulus was fairly appreciated. Dragged pin-point became very sharp at boundary of light touch loss. Single pricks at 27.2 grammes not felt on the outer leg and sole; but rapidly repeated pricks (summation) caused lively sensation of pain with typical protopathic characteristics. (b) *Heat* at 65° C. evoked no sensation below level of light touch loss. For contact slightly longer than momentary, sensibility present, with protopathic characteristics, for hurt element; present also for warmth element (trace) on the outer leg and sole. Upon thighs sensibility present, chiefly for hurt element, and without abnormal reaction. (c) *Cold* (ice) felt as "hurt" on the outer leg and sole, and as "hot" and burning from level of light touch loss up to level of tenth thoracic root where specific element of cold began to be appreciated. Protopathic characteristics on lower abdomen and limbs.

III. DEEP CRITICAL. (a) *Pressure-touch.* At 2.3 grammes sensibility absent to knees (root LIII). Threshold average on middle of outer leg at 6.7 grammes; and at 9.1 grammes on lower outer leg and on foot. (b) *Posture and passive movement.* Good for knees and ankles; improvement in toes. Movement of limbs evoked sharp pains. (c) *Weight.* On dorsum of fully supported foot 200 grammes not felt, but on outer leg the weight felt as "something"; above knee it was felt clearly. Legs and feet extremely tender;

spontaneous pains. (d) *Compass points*, at $3\frac{1}{2}$ inches separation, applied consecutively and longitudinally, well appreciated above knee. On outer leg and dorsum of foot appreciated only at times.

IV. DEEP AFFECTIVE. (a) *Pressure-pain*. Algometer threshold averages: Outer dorsum foot at 0.5 kilo; middle thigh front, at 1.0 kilo; outer aspect middle thigh at 1.5 kilos; outer aspect middle leg at 0.5 kilo. In various regions tested protopathic characteristics in evidence but less so than on previous occasions, and more so on foot and outer leg than on thighs. (b) *Heat*. In large jug, with firm, more or less prolonged application 65° C. was felt hot (warm) on outer leg and sole. Entry into consciousness not so sudden, and sensation not so unpleasant as on previous occasions. Less spreading; greater control. (c) *Cold*. In a large jug 10° C. felt cool on sole and outer leg. Protopathic characteristics less than on previous occasions. (d) *Vibration*. Present with associated hurt element, all over lower limbs; radiation on thighs, legs and feet. Hurt element attributed mainly to tenderness of skin of leg and foot (superficial affective), as "heel" of tuning fork was slightly angular. Radiation on thighs, etc., indicated returned function rather than a protopathic characteristic as normally vibration radiates considerably.

Summary. Tenth day: 1. *Mentality and Motor*. About as on fifth and sixth days. 2. *Sensory*. A. *Spontaneous*. Shooting pains in legs and feet; to less extent, in thighs. B. *Elicited*. I. SUPERFICIAL CRITICAL. Absent for light *touch* and *warmth* up to root L IV; and for *cold* (critical element) up to root thoracic X. II. SUPERFICIAL AFFECTIVE. Absent for *single pricks* at 3.0 grammes and for 65° C. (momentary application) up to level of root L IV; present, with protopathic characteristics, upon legs and feet for hurt element of *repeated pricking*, of 65° C. (slightly prolonged application) and of ice. Latter left as burning on thighs; not felt as cold up to the root thoracic X. II. DEEP CRITICAL. Marked improvement in legs and feet. IV. DEEP AFFECTIVE. Recession of protopathic characteristics.

By way of contrast the sensory condition as found on various days, before and after operation, may be set down as follows:

	<i>Before operation</i>		<i>After operation</i>	
	Aug. 10	2nd day	5th day	10th day
Superficial critical	0	0	0	+ ♀
Superficial affective:				
Hurt element	0	0	++	+
Specific critical elements	0	0	0	+
Protopathic characteristics	0	0	+	+ ♀
Deep critical	0	0	+	++
Deep affective:				
Hurt element	+	++	+	+ ♀
Specific critical elements	0	0	+	++
Protopathic characteristics	+	++	+ ♀	+ ♀ ♀

Thirteenth day. 1. *Mentality.* Good.

2. *Motor.* Spontaneous jerky movements of legs which set in on eleventh day; spontaneous bladder evacuations though patient has some control.

3. *Sensory.* A. *Spontaneous.* Pains, chiefly in thighs, but also in legs and feet; intermittent, short lived, jumping from place to place; burning and itching in legs and feet, chiefly in feet; patient wants to have feet rubbed. B. *Elicited.* I. SUPERFICIAL CRITICAL. (a) *Light touch.* Absent for 0.5 gramme von Frey hair ventrally and laterally to just above root L III; absent dorsally on thighs excepting area $1\frac{1}{2}$ inches in diameter just below left gluteal crease. (b) For *compass points*, at three inches separation, applied simultaneously and longitudinally, sensibility fair on middle thigh ventrally; over legs and feet absolute inability to distinguish "twos" from "ones."

II. SUPERFICIAL AFFECTIVE. (a) For *single pricks* at 3.0 grammes absent up to middle thigh ventrally (root L II); at 8.6 grammes absent to one inch below boundary of light touch loss. Dragged pin-point became very sharp at boundary of light touch loss. Superficial pinching of skin on outer aspect of middle of leg attended by marked protopathic characteristics, *i.e.*, radiation, reference, etc.

III. DEEP CRITICAL. (a) *Pressure-touch.* For 2.3 grammes absent to about one inch below boundary of light touch loss; for 41.7 grammes absent on foot and up to middle of outer aspect of each leg. On dorsum of foot this stimulus evoked the flexion reflex, the movement of the limb causing pain with subjective over-reaction (exclamation, etc.). (b) *Posture and passive movement.* Fair for hips, knees, ankles and toes; every movement caused pain. (c) For *compass points* at three inches separation, consecutively and longitudinally applied, sensibility fair. (d) *Weight.* Could not, at times, appreciate increment or decrement for 100 grammes added to, or taken from, 100 grammes resting upon the mid-peroneal aspect of leg.

IV. DEEP AFFECTIVE. (a) *Pressure-pain.* Algometer threshold average on outer aspect of middle of leg at 3.5 kilos; trace of protopathic characteristics.

Summary. Thirteenth day. Spontaneous pains in the legs and feet; recession of critical sensibility, chiefly of superficial forms, and return of some of protopathic features attending superficial affective stimulation.

Death of patient. On the tenth day the bed-sore over the sacrum, which had been doing well under treatment, took a turn for the worse. The temperature rose to 102.5° F. With this came a somewhat sudden regression of whatever function had been regained since the operation. The sore sloughed; the patient sank gradually and died from exhaustion on the thirty-third day.

CASE IV. EXTRADURAL NEOPLASM CAUSING PARAPLEGIA IN FLEXION WITH PROGRESSIVE ABOLITION OF SENSIBILITY IN LOWER LIMBS, CRITICAL ELEMENTS BEING FIRST, AND DEEP AFFECTIVE LAST TO DISAPPEAR. AS DEEP CRITICAL ELEMENTS BECAME SUPPRESSED DEEP AFFECTIVE ELEMENTS TOOK ON PROTOPATHIC CHARACTERISTICS. AFTER TOTAL ABOLITION OF SENSATION, THE SPINAL REFLEXES, *e.g.*, FLEXION REFLEX, WERE READILY ELICITED BY AFFECTIVE STIMULATION, THUS SHOWING THAT ABOLITION OF SENSATION WAS RESULT OF CORD AND NOT OF SPINAL ROOT COMPRESSION.

S. M., female, forty-one years. Onset December, 1913. Woke up with pains about waist. These disappeared on exercise but returned at intervals. Pains present especially on awaking and confined to waist. At first intermittent, pains gradually became more persistent and severe with occasional remissions. Seven months after onset *right leg* "gave way" in walking and in four days became useless. As leg grew weak sensations of heat and cold (paraesthesiae) appeared in it and the foot and ankle became swollen. Eleven days after appearance of weakness in right leg remittent pains appeared in toes of *left foot*. These extended to leg and knee as limb became weak and stiffened. By January 1, 1916, patient had lost all control of both lower limbs. On March 4, 1916, an operation was performed in Bellevue Hospital by Drs. Lucius W. Hotchkiss and Harold Neuhof. Operative scar extends from the second to the eighth thoracic spinous process. After operation patient's condition not appreciably improved. Entered the Central Neurological Hospital on Welfare Island where she was studied by the author at frequent intervals from December 19 to July 17, 1917. Examination, after catheterization, December 19 and 21, 1916.

1. *Mentality*. Keen; patient alert and coöperative.

2. *Motor*. Slight convergence of eyes on looking up; lower abdominal muscles flabby; spastic paralysis of both lower limbs. Spontaneous flexion of hips, knees and ankles (dorsal) more marked on right side. No fibrillation or other abnormal movements. As one limb flexes the other extends more or less actively. Slight hypotonia of calf muscles. Right and left calf $12\frac{1}{2}$ inches. Urine and feces retained, necessitating regular catheterization and enemata.

3. *Sensory*. A. *Spontaneous*. Zone of pain about waist at level of thoracic roots VIII; pains also in right knee and in region of psoasiliac muscle when knee flexes; pain and paraesthesiae (burning, pins and needles, formication) in both lower extremities, more especially on feet and upon outer aspect of thighs and legs; extremities feel cold to examiner's hand. B. *Elicited*. I. SUPERFICIAL CRITICAL. (a) *Light touch*. For the 0.5 gramme von Frey hair, absent to root thoracic VIII on right and thoracic IX on left. Chart 7. (b) *Warmth*. For 38° C., absent to root thoracic X on both sides. (c) *Cold*. For 25° C., about as for 38° C. (d) *Compass points*. Gross impairment below level of light touch loss.

II. SUPERFICIAL AFFECTIVE. (a) For *single pricks*, at 3.0 grammes, absent for critical element (pointedness) to just below lower boundary of light touch loss; present for hurt element on areas of linear shading in Chart 7. On right foot stimulus evoked flexion reflex and a very unpleasant, poorly localized sensation which was attended by radiation reference and inability to name the stimulus. Pricking, at 13.4 grammes, absent for critical element (pointedness) on right side to $2\frac{1}{4}$ inches, and on left to 1 inch, below boundary of loss for pricking at 3.0 grammes; present for hurt element on feet, legs, and lower part of thighs, with typical protopathic characteristics. Subjective over-reaction, though present at times, was never marked. Introspective evaluation for pricking at 13.4 grammes on lower leg and dorsum of foot:

	Normal	Affected area
Touch.....	+	0
Pointedness	+++	0
Hurt	+	++
Subjective over-reaction	0	+(trace at times)
Objective over-reaction	0	++
Localization	+	0
Radiation and reference.....	0	++
Ability to name the stimulus.....	+	0

For dragged pin-point zone of hyperalgesia $1\frac{1}{2}$ inches wide on trunk about lower limit of preserved (impaired) sensibility for light touch. In areas of linear shading in Chart 7, hyperalgesia also for dragged pin-point. (b) *Heat*. For 55° to 60° C., absent for critical element to just below loss for 38° C.; present (delayed) for hurt element, with protopathic characteristics, on legs and feet. Readily evoked flexion when applied to legs, feet and lower thighs. (c) *Cold*. For ice, absent for critical element to one inch below loss for 55° to 60° C.; present (delayed) for affective (hurt) element, with protopathic characteristics, over legs and feet. On soles response prompt *subjectively* and *objectively* compared with response for 55° – 60° C. (d) *Stroking hair* over right leg and foot at times evoked flexion reflex in addition to a spreading, creeping, unpleasant sensation like a needle prick. (e) *Pulling single hairs*, on right leg and foot, at times evoked a disagreeable, spreading sensation, and flexion reflex. Introspective evaluation for 55° – 60° C. and 0° C. on lower leg and foot:

	Normal	Affected area
Touch	+	0
Warmth, cold	+++	0
Hurt, unpleasant	+	+++
Subjective over-reaction	0 (or trace)	+
Objective over-reaction	0 (or trace)	+++
Localization	+	0
Reference	0 (or trace)	+
Radiation	0 (or trace)	+
Ability to name stimulus	+	0

III. DEEP CRITICAL. (a) *Pressure-touch*. For 2.3 grammes absent for purely critical element to root thoracic X. For 41.7 grammes to root thoracic XI. On foot and lower leg, especially on right, heavier pressures evoked an unpleasant, creeping, spreading sensation and flexion reflex. (b) *Posture and passive movement*. Absent for all ranges at knees, ankles, and toes; grossly impaired at hips for movement through an arc exceeding 20 degrees. (c) *Compass points*. Absent at 4 to 6 inches separation, applied consecutively and longitudinally, upon thighs, legs, and feet. (d) Tests for *size, shape, weight, roughness* (critical element), *consistency, texture, etc.*, not made.

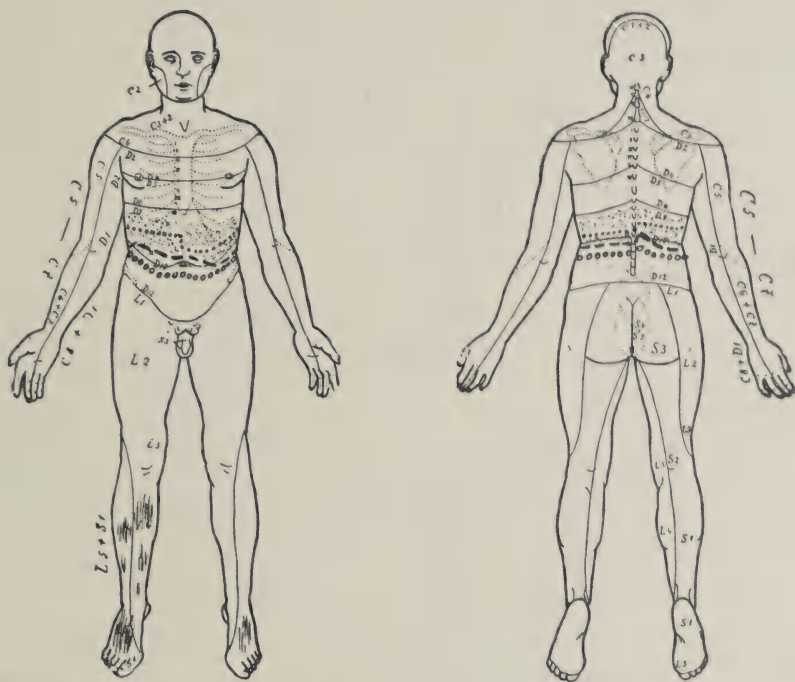


CHART 7. Case 4. Extradural neoplasm. Protopathic dissociation from remote cord compression. *Light stippling*: impaired sensibility for 0.5 gramme von Frey hair. *Heavy dots*: bound loss for cotton wool. *Dashes*: bound loss for pricking at 3.0 grammes. *Circles*: bound loss for pressure-touch at 2.3 grammes. *Linear shading*: superficial affective sensibility retained with protopathic characteristics.

IV. DEEP AFFECTIVE. (a) *Pressure-pain*. Algometer threshold averages: (1) *Ball of great toe*. Right side: flexion reflex at 0.5 kilo and at 1.0 kilo pain, attended by spreading (to the foot and leg) and reference (to back). Left side: flexion reflex at 1.5 kilos and at 5.0 kilos pain, attended by spreading (to the leg and foot) and reference (to back). (2) *Anterior tibial region*. Right side: flexion reflex at 1.5 kilos and at 5.0 kilos pain, with spreading (to the thigh

and abdomen) and reference (to back). Left side: flexion reflex at 5.0 kilos with no pain for as high as 10.0 kilos, greater pressures not being tried. (3) *Gluteal region*. Right side: flexion reflex at 8.0 kilos and pain at 11.0 kilos. Left side: flexion reflex and pain, attended by over-reaction, etc., appeared simultaneously at 5.5 kilos. (4) *Pinching* skin deeply over legs, feet, and lower thighs, caused poorly localized pain with radiation. (b) *Heat*. Water at 55°–60° C. in large jug, upon moderately prolonged, firm application to lower thighs, legs and feet elicited flexion reflex and, especially when applied to legs and feet, hurt, like pin-prick, with protopathic characteristics. Subjective over-reaction absent below boundary of loss for pricking at 16.4 grammes. Critical element absent to root thoracic X. (c) *Cold*. Present (delayed), with protopathic characteristics, for hurt element of ice in large jug, on thighs, legs and feet. Marked over-reaction subjective and objective. Absent for critical element up to one inch below level of loss for 55°–60° C. in massive application. Evaluation for 55°–60° C. and ice in massive application:

	Normal	Patient	
Touch	+	0	
Warmth, cold	+++	0	
Hurt, unpleasant	+	++	
Subjective over-reaction..	0 (or trace)	+	
Objective over-reaction...	0 (or trace)	+++	(For ice, absent for
Localization	+	0	55° to 60° C.)
Radiation	0 (or trace)	++	
Reference	0	++	
Ability to name stimulus..	+	0	

(d) *Vibration*. Present for affective element (quality of sensation) on dorsum and sole of each foot. Rate of vibration (critical element) considerably slowed as compared with hands. Absent over rest of lower limbs for all elements. (e) *Roughness*. Present, upon feet and lower legs, for affective element but readiness with which flexion reflex appeared made introspective analysis unprofitable.

4. *Reflexes*.

	Right	Left
Epigastric	0	0
Abdominal	0	0
Anal	+ ♀	+
Elbow	++	++
Wrist	++	++
Knee (limbs flexed, patient lying down)	0	0
Ankle	+ ♀ ♀	+ ♀ ♀
Ankle clonus (knee flexed, etc.)	0	0
Plantar	A, ♂ (flexion reflex)	H, ♂ ; R, O
Oppenheim	A, ♂ (flexion reflex)	A, ♂
Gordon	A, ♂ (flexion reflex)	A, ♂ (flexion reflex)

Under certain conditions, *e.g.*, patient sitting up and legs hanging over edge of bed, or when opposite limb was in phase of active flexion, the knee jerks were present but markedly impaired, more especially in left leg where a trace of muscle movement was barely

visible. Similarly under certain conditions, *e.g.*, with leg fully extended on bed, *i.e.*, with the flexors (calf muscles) in moderate tension, true ankle clonus was present, *i.e.*, with foot at a right or acute angle with the tibia. The receptive field for the flexion reflex was considerably widened the reflex being readily elicited by stroking, with blunt and sharp instruments, the foot and lower leg as well as by pinching and pricking all over foot, leg, and lower half of thigh.

From January to July, 1917, the disease progressed. The level of lost sensibility ascended to the nipple region, root thoracic V. Examination July 17, 1917:

1. *Mentality.* Good.

2. *Motor.* As in January. Legs continue to "draw up."

3. *Sensory.* A. *Spontaneous.* Pain referred to outer part of legs when the latter "draw up." B. *Elicited.* Absent for all degrees and forms of critical and affective stimulation in lower limbs and on trunk below umbilicus. On thighs, legs, abdomen, and at times on the perineum, affective stimuli of all sorts and degrees elicited the flexion reflex. Extreme pressure on calves, severe pinching of skin, and hair pulling over legs, feet, and thighs, as well as extremes of heat and cold (70° C. and ice) in massive application failed to evoke any sensation whatever although each of these stimuli promptly elicited the flexion reflex.

4. *Reflexes.* About as on December 19, 1916. The receptive field for the flexion reflex had evidently been widened to include the upper thigh, perineum and part of the abdomen.

General Summary. 1. *Motor.* Typical paraplegia in flexion from compression that functionally isolated portion of cord below segment LI.

2. *Sensory.* Gradual abolition in order for these stimuli (a) *superficial critical*; (b) *deep critical*; (c) *superficial affective*; (d) *deep affective*. Where sensibility for the affective (hurt) element was retained after suppression of the corresponding critical elements it always exhibited protopathic characteristics which, with the exception of subjective over-reaction, were very pronounced.

3. *Reflexes.* These in the main were characteristic for the type of cord lesion, *viz.*, complete functional interruption. The patency of the afferent and efferent arcs serving the flexion reflex, etc., was significant as clearly showing that the compression causing the protopathic dissociation in the earlier stages, and complete loss of sensibility in the later stages, was exerted not upon the lumbar nerve-roots but upon the spinal cord itself at a level well cephalad of the level of the lumbar nerve-roots. After the examination made in July the patient gradually became weak and emaciated and died in August, 1917, of exhaustion.

(To be continued.)

THE THERAPEUTIC APPLICATIONS OF THE EFFECT OF HYPERTONIC SOLUTIONS ON THE CEREBRO-SPINAL FLUID PRESSURE: A CRITICAL REVIEW *

BY H. C. SOLOMON, M.D.

CHIEF OF THERAPEUTIC RESEARCH, BOSTON PSYCHOPATHIC HOSPITAL

L. J. THOMPSON, M.D.

LATELY SENIOR MEDICAL OFFICER, BOSTON PSYCHOPATHIC HOSPITAL

H. M. PFEIFFER, M.D.

EXTERNE, BOSTON PSYCHOPATHIC HOSPITAL

The work of Weed and McKibben,(1) showing that the introduction of hypertonic saline solution into the circulation will cause a drop in the cerebrospinal fluid pressure, has led to attempts to utilize this phenomenon in the treatment of diseases of the central nervous system, notably neurosyphilis. The purpose of this communication is to discuss these possibilities.

It seems advisable to review briefly the findings of Weed and McKibben. They showed in their experiments on cats that the introduction of a hypertonic solution of sodium chloride caused a pronounced drop in the cerebrospinal fluid pressure, which fell from its original pressure to zero, or in many cases, to a definite negative pressure. They conclude that this drop in pressure is the result of the body's attempt to draw fluid from all available sources into the blood stream in order to reestablish the normal osmotic pressure and reduce the concentration of the chlorides in the blood. The reduction of the spinal fluid pressure in these cases is not due to a change in blood pressure, which does not correspond by any means with the drop in fluid pressure. In confirmation of the idea of Weed and McKibben that the fluid is drawn into the blood, Barach, Mason and Jones (2) have shown that there is an increase of the blood volume following the introduction of hypertonic saline, and that this increase in blood volume is due almost entirely to increase in the plasma volume.

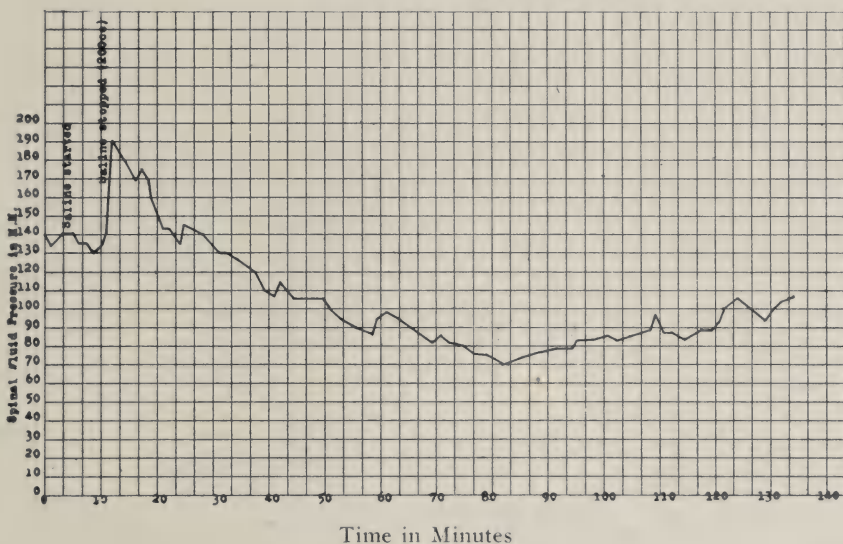
Weed and McKibben state that a maximum drop in fluid pressure in their experimental animals occurred in from 25 to 30 minutes on the average, and a short period thereafter the pressure again began to rise. Barach, Mason and Jones (2) showed that the gradual

* From the Boston Psychopathic Hospital.

return to normal blood volume began a short time after its rapid increase and that the normal was reached within three and a half hours after the injection. This agrees with the finding that the spinal fluid pressure tends to rise after a short period of time, apparently indicating that fluid is no longer necessary to dilute the blood and hence newly formed cerebrospinal fluid is utilized to produce an increase in the spinal fluid pressure. On the introduction intra-venously of the hypertonic saline, there is an immediate rise in spinal fluid pressure, which rise continues during part of the period taken

CHART I

Effect on Spinal Fluid Pressure of the Intravenous Injection of 200 c.c. of 15 per cent NaCl



in the introduction of the saline and is followed by a notable fall in the pressure. Weed and Hughson (3) consider this initial rise to be the result of venous pressure changes which are of short duration. The appended Chart 1 shows the results graphically, the form of the curve being similar in man and in the cats used experimentally. Weed and McKibben, (1) and Foley and Putnam (4) have given evidence to show that the egress of fluid from the cerebrospinal fluid spaces takes place through the choroid plexus and the perivascular and perineuronal spaces.

To summarize these results the following may be stated: With the introduction of hypertonic solution of sodium chloride there

occurs a brief rise in spinal fluid pressure, followed by a fall to a point below the original level, after which there is again a rise of the cerebrospinal fluid toward normal. The drop is apparently due to the removal of fluid from the cerebrospinal fluid system into the blood stream for the purpose of reducing the concentration of saline. The outflow is through the choroid plexus and perivascular and perineuronal spaces. The results in man are entirely similar to those obtained in animals, as has been shown by Foley and Putnam,(4) Wynn,(5) Ebaugh and Stevenson,(6) ourselves and others, and the charts which we include give a definite picture of this phenomenon.

Further evidence of the fall of intracranial pressure is shown by the effect of hypertonic solutions in cases of herniated brains in brain tumor cases that have been decompressed. The herniated brain mass may be seen to retract for several hours after the injection but when the effects of the salt have worn off, the brain again herniates.

Foley suggests that the tendency of the cerebrospinal fluid to pass through the choroid plexus and through the perivascular spaces of the nervous system after the injection of hypertonic solutions, might be utilized to carry medicaments introduced into the subarachnoid space into the tissue of the nervous system. Wynn (5) followed this suggestion in the treatment of cases of neurosyphilis, particularly tabes, in the following manner: An intraspinal injection of serum was given into the lumbar subarachnoid space and this was followed by an intravenous injection of 200 c.c. of a 15 per cent sodium chloride solution, the theory, as already suggested, being that the serum would follow the route of the cerebrospinal fluid. The logic of this procedure seems correct, and the result, which will be considered shortly, seems to bear out the logic.

Corbus, Lincoln, O'Connor and Gardner (7) suggest another application of the fall of spinal fluid pressure produced by the intravenous injection of hypertonic salt solution. They argue that following the fall of cerebrospinal fluid pressure there again occurs a rise of fluid pressure. This they assume is produced by an increased secretion of cerebrospinal fluid, *i.e.*, that the choroid plexus is stimulated to increased secretion in order to reestablish the normal spinal fluid pressure. Their further argument is that if there is an increased secretion of cerebrospinal fluid, an introduction of medicament into the blood stream during the period of increased secretion will give the optimum conditions for entrance of such medicaments through the choroid plexus or perivascularly. Their utilization of these assumptions in the treatment of neurosyphilis is to give the patient an injection of 100 c.c. of 15 per cent sodium chloride solution fol-

lowed in six hours by an injection of arsphenamine. The point made is that six hours after the introduction of the sodium chloride the secretion of the choroid plexus will be greater than normal and that there is a greater chance, therefore, of arsenic passing through the choroid plexus and reaching the central nervous system and the cerebrospinal fluid. They have given this procedure the euphemistic title, "Spinal Drainage Without Lumbar Puncture. A New Method for Increasing the Penetration of Arsenic into the Spinal Fluid in the Treatment of Neurosyphilis."

It is our purpose to give a critical review of these two ideas of Wynn, and Corbus *et al*, on the basis of our own experimental work and subject their experiments to as severe criticism as we are able.

We will first study the work of Wynn.(5) As already noted, we believe the logic of the procedure to be quite sound, and our consideration, therefore, will be based largely upon the clinical value of this procedure. In the first place we must consider the effect of the introduction of relatively concentrated solutions of sodium chloride. What we have to say in this regard has a bearing upon our criticism of the method of Corbus *et al*, as well as upon that of Wynn. Fifteen per cent solution is one that has been chosen by Foley and Morris (8) and Wynn (5) as about the most satisfactory concentration for intravenous injection in man. Weed and McKibben originally showed that the effect on the cerebrospinal fluid pressure varied within limits with the concentration of the solution. The greater the concentration, the more the fall in pressure. Foley and Morris (8) point out that relatively large drops are obtained from a concentration of 15 per cent, whereas the higher concentration does not give enough difference in effect to compensate for the increased toxicity. Wynn (5) has followed the procedure of using 200 c.c. of 15 per cent sodium chloride. Now, the effect of intravenous injection of 15 per cent sodium chloride is very uncomfortable as is borne out by Wynn's statement and our own experience. Almost immediately upon the introduction of this solution respiration becomes labored, the patient becomes flushed, and complains of dryness of the mouth, very unpleasant burning sensations, and severe headache.

In a series of sixteen patients treated by Wynn, he lists the results as "very distressing" in eleven; "slight distress" in four; and "no distress" in one. He states, "Almost every patient experienced more or less marked discomfort from the hypertonic solutions, in six cases this discomfort being severe enough to necessitate abandoning subsequent injections." He found that the severe headache,

although it never lasted more than from fifteen to twenty minutes, could be controlled by nothing but morphine.

Weed and McKibben (1) in their early report call attention to the toxicity of sodium salts in their animals and the following may be quoted:

"It was early noticed that the intravenous injection of the concentrated solutions of sodium chloride was followed by severe respiratory and cardiac disturbances. It was often necessary to alter the anesthetic to avoid losing the animal. The ether was in these cases reduced and artificial respiration used, the intratracheal method of anesthesia making possible a nearly ideal artificial respiration. This toxicity of the sodium chloride was apparently not dependent upon the absolute amount injected but was frequently observed in the earliest intervals of the injection when only 1 to 2 c.c. had been introduced. As soon as this initial toxicity was passed, further injection up to certain limits could be made with comparative safety in the same animal. Such early disturbances seem best explained on the basis of an alteration in the balance of the mineral salts in the blood.

"In addition to the initial toxicity of the sodium chloride, another factor of importance in these experiments was variation in the tolerance of the individual animal. Some animals, showing no apparent disturbance on receiving relatively large intravenous injections, have given marked reactions as indicated by a lowering of the pressure of the cerebrospinal fluid. In still other animals where the toxic effects of the salt were marked, the lowering of the pressure of the cerebrospinal fluid was definite and considerable. Thus an individual tolerance and reaction for each particular animal seems indicated, a tolerance and reaction which we have not been able to predict in our experiments."

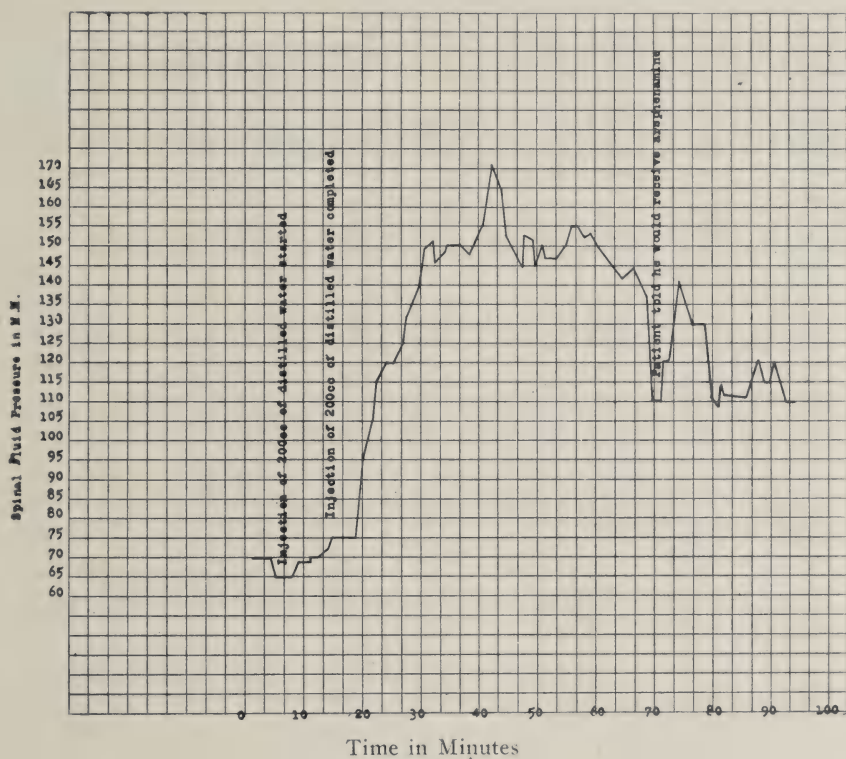
In a later article Weed and Hughson (3) review the toxic effects of sodium chloride injections and from their paper the following may be quoted:

"The general systemic effects of the intravenous injection of hypertonic solutions, apart from the pressure of the cerebrospinal fluid, have been investigated by many workers. The first contribution to the subject was apparently that made by Guttman, who showed that 5 grams of sodium chloride injected as a 20 per cent solution caused the death of rabbits. Klikowicz determined the chemical changes in the blood occurring after intravenous injection of sodium chloride in dogs. A marked increase (roughly 50 per cent) in the chlorides of both whole blood and serum was found in specimens obtained two minutes after the injection; after one hour the chloride content was still high. Heinecke studied the reactions of frogs to immersion in strongly hypertonic solutions of sodium chloride; prolonged or repeated immersion led to convulsions and death. In an investigation of the effect of sodium chloride in large doses upon intravascular clotting, Silbermann found that the intravenous injection of 4 to 12 grams of sodium chloride, inevitably led to convul-

sions and death in the rabbit. Heinz was apparently the first to observe blood pressure changes after injection of hypertonic sodium chloride solutions; arterial pressure after injection of 20 c.c. of a concentrated solution of sodium chloride (5 per cent) was found to fall gradually until the death of the animal. Münzer recorded a similar fall in carotid pressure from the beginning of the injection

CHART 2

Effect on Spinal Fluid Pressure of the Intravenous Injection of 200 c.c. of Distilled Water



of a 10 per cent solution of sodium chloride; this decrease in pressure continued to death, rising during the convulsions. The lethal toxic dose of sodium chloride was determined by Münzer to be 3.72 grams per kilogram of body weight, if given as a 10 per cent solution. Selig, studying the effect of inorganic salts upon lowered blood pressure, found that the intravenous injection of 1.8 per cent solution of sodium chloride raised the pressure more efficiently than a like injection of an isotonic solution. Similar conclusions that hypertonic solutions (5 to 10 per cent) of sodium chloride in small doses were capable of raising the arterial pressures in exsanguinated animals

were reached by Retzlaff. In an important study of this question, Seppä found that the injection of a hypertonic solution (sodium chloride) caused a sudden increase in the arterial pressure, due apparently to contraction of the blood vessels; this was followed in 30 to 70 seconds by a fall to the initial level. Then more slowly there occurred a secondary increase in arterial pressure due to the fluid flowing from the tissues into the blood stream with later a gradual recession from this high point of pressure. A difference in lethal dosage of such solutions of sodium chloride was noted by Seppä in intact and exsanguinated animals; the lethal dosage varied from 1.70 to 1.95 grams per kilogram for the intact animal and 0.198 to 0.29 grams per kilogram of body weight for the bled. On rapid injection (1 c.c. per minute) of 26.4 per cent solution of sodium chloride, Seppä ascertained the lethal dosage to be 1.95 grams per kilogram of animal—a lower value than that of previous investigators. He attributed the lowering of the lethal amount of salt to the strength of the injection-fluid and the rapidity with which the injections were given.”

It will be seen from these statements and from the experience with human patients that the process of hypertonic salt solution injections is one that should not be entered into haphazard. With the dosage recommended by Foley and Morris (8) and utilized by Wynn, (5) Corbus *et al*, (7) and ourselves, no serious results have been obtained. Nevertheless, it must be recognized that the process is one which causes a great deal of discomfort and inconvenience to the patient. It is interesting to note that the effects on man are similar to those upon animals in so far as that, with the introduction of a very small amount of this solution, unpleasant symptoms occur almost immediately. Therefore, reducing the dose does not avoid these symptoms, although it may keep one well within the realm of safety as far as life is concerned.

Another factor that militates against the value of this procedure is the effect upon the vein used for injection. A concentrated salt solution causes sclerosis of the wall of the vein and after one or two injections it is usually impossible to use this vein again for intravenous injections. This is a matter of considerable consequence in the treatment of neurosyphilis which is likely to require numerous intravenous injections. In the technique of Wynn, (5) in addition to the unpleasant effects of chloride injections, one must also consider the effect of the combination of intraspinal and hypertonic solution injections. As already stated, there is some evidence that experience bears out Foley's idea of increased movement of the serum as produced by the hypertonic salt injection. The patients almost invariably have great discomfort and pain as a result of the intraspinal injection. This

pain is very much more marked than that which usually occurs in intraspinal therapy in cases of neurosyphilis, although in tabes it is often quite severe. However, in our experience, and we take it also in that of Wynn, the pain is so greatly increased as to become almost intolerable and hardly controllable by opiates. As noted, this would seem to bear out the hypothesis of Foley. Nevertheless, the discomfort becomes so great as to make a definite contraindication.

In summarizing the conclusions of his experiments, Wynn (5) makes the following statement, "The cases treated showed no serologic or cytologic improvement over the usual course with intraspinal treatment alone." From the clinical and serological standpoint, therefore, it is fair to conclude that this method has little value over that of the usual method of intraspinal therapy, and when we consider in addition the unpleasant factors, namely the discomfort and distress produced by the intravenous injection of hypertonic solutions, the increased pain of the intraspinal treatment, and the tendency of the injection to sclerose the vein, it would seem justifiable to conclude that this method is not practical in the treatment of neurosyphilis.

The correctness of the theory on which Corbus and his co-workers base their work is not entirely certain and merits some consideration. Their technique consists of the introduction of a 100 c.c. of a 15 per cent sodium chloride solution, followed in six hours by 0.9 of a gram of neoarsphenamine. This time relation is based on Foley's observations of the cases of decompressed hernia, where he found that on the introduction of the hypertonic saline solution, the hernia gradually receded for two or three hours and the return of the herniation began from six to ten hours after the injection. On this basis Corbus and his co-workers assume that there is an increased secretion of cerebrospinal fluid beginning in the neighborhood of six hours after the introduction of the saline solution. We should like to point out that this is as yet an unproven assumption.

While there seems to be sufficient evidence to show that with the introduction of hypertonic saline solution there is increased absorption of cerebrospinal fluid from the ordinary fluid channels, we know little or nothing concerning the rate of formation of fluid during this period, nor, as a matter of fact, do we know anything very definite about the rate of formation of spinal fluid under more normal circumstances. It is indeed possible that the return of brain bulk or fluid in the fluid system is produced by the normal rate of secretion of the fluid with less absorption rather than by increased secretion.

From the work of Barach *et al.*, (2) it is to be concluded that the

increased outflow of cerebrospinal fluid takes place within a relatively short period, as the increase in blood volume continues only for a half hour or thereabouts, and likewise the drop in fluid pressure ceases after some such time.

It has been estimated, whether correctly or not, that under ordinary circumstances the cerebrospinal fluid renews itself three or four times during twenty-four hours. If this is so then with a slightly reduced absorption of cerebrospinal fluid the pressure would return to normal after some hours without an increased rate of secretion. Further, in their animal experiments, Foley and Putnam (4) have shown that the return to normal pressure is really very slow. For instance, in a fairly typical experiment in one animal, a drop from 240 mm. of spinal fluid pressure to —40 mm. was obtained and at the end of nine hours the fluid pressure had risen only to 145 mm., or 95 mm. below what it was at the start of the experiment. Similarly,

CHART 3

Ex- peri- ment	Original Pressure	Amt. of Fluid Withdrawn	Pressure after Withdrawal	More Fluid Withdrawn	Pressure after 2nd Withdrawal	More Fluid Withdrawn	Pressure after 3rd Withdrawal
1	60 mm.	5 cc.	42 mm.				
2	70 mm.	10 cc.	40 mm.				
3	130 mm.	10 cc.	60 mm.				
4	145 mm.	15 cc.	110 mm.	20 cc.	70 mm.	20 cc.	32 mm.
5	160 mm.	25 cc.	70 mm.	25 cc.	10 mm.		
6	160 mm.	26 cc.	0 mm.				
7	60 mm.	30 cc.	0 mm.				
8	120 mm.	35 cc.	0 mm.				
9	130 mm.	40 cc.	0 mm.				
10	110 mm.	50 cc.	0 mm.				
11	195 mm.	50 cc.	0 mm.				

in the group of experiments, they found that at the end of 17 to 48 hours the pressure had not returned to normal. While these latter experiments in which the dura and arachnoid were punctured are not entirely comparable to those in which no puncture is made, nevertheless we have, as yet, no proof that following the injection of salt there occurs later an increased secretion of cerebrospinal fluid. But, for the purpose of further evaluation of this therapeutic procedure let us assume that there is some increased rate of cerebrospinal fluid formation as a result of the body's attempt to reconstitute the pressure conditions that should normally exist.

The literature is particularly free of any data concerning the fluid pressure for any considerable period of time after the injection of hypertonic saline solution. Corbus and his co-workers apparently accepted the observations of Foley on the change in brain bulk in cases of intracranial pressure in an animal as being quite the same as what

would occur in man. In the case of the herniated brain the conditions are quite different from those occurring normally, and the results, therefore, cannot be expected to be entirely similar. Thus, as will be shown below, the assumption concerning the similarity between animal experiments and man, while probably justified, has to be modified on account of the difference in dosage.

Wynn,(5) in his work, followed the spinal fluid pressure in six cases for about thirty minutes after the intravenous injection of the salt solution. He states that in the six cases studied, there was a constant rise in the pressure of the cerebrospinal fluid of 30 to 50 mm during the fifteen minutes occupied in giving the solution, after which the pressure fell so that by thirty minutes after the end of the injection it was usually 80 to 100 mm, below the original level. He continues. "However, the close correspondence in these initial pressure variations with those in the cat would make it reasonable to suppose that the depression increases till zero and possibly even negative pressures are reached." Now, this assumption is not justified. As already noted, there is a variation in the degree of pressure drop dependent upon the amount of saline injected. In the article by Weed and Hughson,(3) it was stated that the average reduction in pressure reported by them was produced by a solution containing about 1 gram of sodium chloride per kilogram of body weight. In the case of the technique employed by Wynn, 200 c.c. of 15 per cent solution were given which would mean 30 grams of sodium chloride given to an individual of about 70 kilograms in weight. This would be less than a half gram per kilogram of body weight, so that it might not produce as marked results as Weed obtained in his animal experiments. Weed and McKibben (1) make the following statement: "As soon as the injection is completed, the pressure of the cerebrospinal fluid starts to fall and a rapid lowering is recorded during the next few minutes. The rate of fall then decreases shortly before the pressure reaches its lowest point. The maximum effect of the intravenous injection, *i.e.*, the point of lowest pressure, is usually noted in from fifteen to twenty minutes after the completion of the injection."

It is to be noted that Wynn continued his pressure observations for about thirty minutes and then discontinued. By analogy from the animal experiments, he would probably have reached the lower level of pressure that would occur, and a perusal of his graph will show that the rate of drop was very slight in the last ten minutes. Therefore, it would not seem quite reasonable for us to expect that the drop will continue down to zero or a negative pressure. As a matter of fact we have evidence to show that this does not occur. In

continuing observations for two hours or longer, we have obtained a curve which probably represents the actual condition. We noted, as did Weed and McKibben and others in their animals, and as did Wynn in his observations on man, that there occurs a drop which at first is fairly rapid but then becoming slower after a time reaches its completion and then begins to rise. It may, therefore, be assumed that Wynn, in his observations had at the end of half an hour reached a point of almost maximum drop after which an increase again took place. This is what happened in the cases which we followed for a considerable period (Chart 1). Similarly, in the converse experiments where distilled water is used and a rise in spinal fluid pressure is obtained, this rise lasts for an hour and then the pressure again falls (Chart 2).

The point of this discussion is that by the use of 200 c.c. of 15 per cent sodium chloride solution one gets a drop of not more than 50 per cent of the original spinal fluid pressure. In using 100 c.c. of 15 per cent saline according to the technique of Corbus, the drop will be much less. *How much fluid need be withdrawn by lumbar puncture in order to produce a drop of fluid pressure to approximately 50 per cent of the original pressure?* Our investigations on this point show that the withdrawal of 10 to 20 c.c. of spinal fluid will often cause a drop of this extent and at times even greater. The following Chart 3 shows the effect on the fluid pressure of withdrawing varying amounts of fluid in several cases.

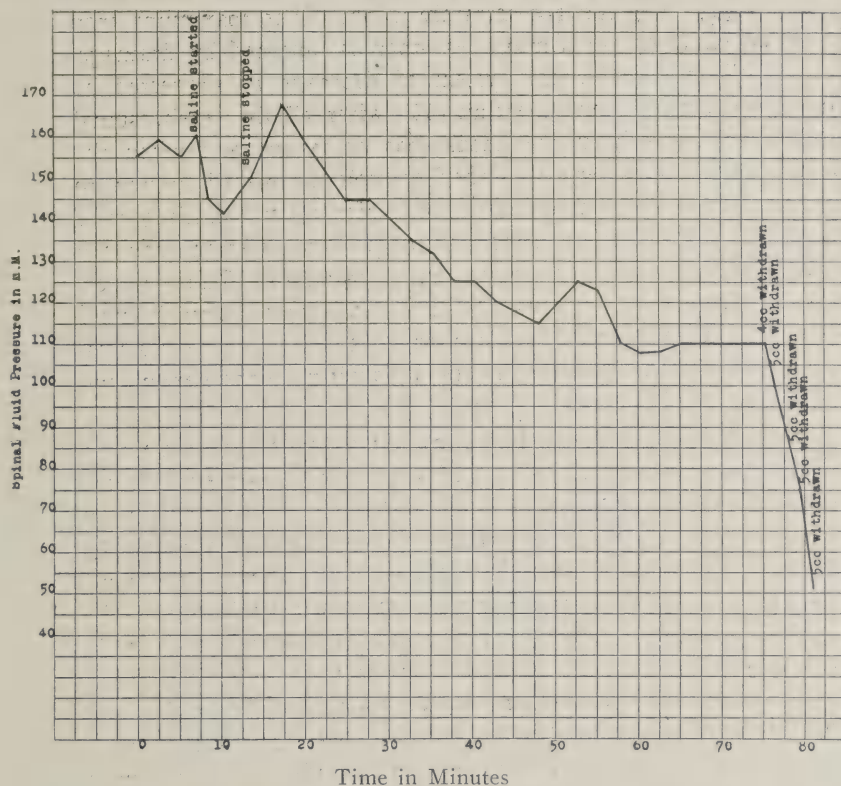
Weston (9) has shown that the ventricles and subarachnoid spaces contain approximately 150 c.c. of cerebrospinal fluid. Hence a drop in spinal fluid pressure of 50 per cent may be produced by a loss of not more than 10 per cent to 20 per cent of the total amount of fluid.

It may therefore be assumed that with the saline injections of 15 grams of sodium chloride the amount of fluid withdrawn from the fluid channels is no greater than in a similar drop produced by spinal drainage, namely, not more than 10 to 20 c.c. If this is so it is not necessary to assume a greatly increased rate of secretion, to compensate for the loss of the small amount of fluid when the compensation occurs in the space of several hours, especially if we assume that the rate of absorption may be decreased. Nor are we certain, by any means, that the increase in pressure which occurs is entirely due to an increase in the amount of fluid. This increase in pressure may be accomplished by changes in the circulation of blood through the brain and possibly also by some elasticity on the part of the dura, especially in its spinal portion.

At any rate, it would seem to be definite that by the technique of Corbus, a very small amount of fluid is withdrawn from the cerebro-spinal fluid system and therefore at the most, there is not a very great increase in the formation of fluid necessary to compensate. This would vitiate the theory on which he bases his procedure.

CHART 4

Effect on Spinal Fluid Pressure of the Intravenous Injection of 200 c.c. of 15 per cent NaCl followed by the Withdrawal of 24 c.c. of Spinal fluid



If we compare the results of drainage by lumbar puncture with the effects of injection of saline, it is at once evident that it is possible to accomplish much more in the way of reduction of cerebro-spinal fluid pressure by puncture drainage than by the method of hypertonic solution injection. The accompanying chart 4 indicates this point. In this experiment 200 c.c. of 15 per cent sodium chloride solution was given intravenously and the spinal fluid pressures as shown by manometric readings were charted. At the end of 45

minutes the pressure had fallen from an original 155 mm. to 110 mm. The pressure then remained stationary at 110 mm. for about 15 minutes, indicating that the maximum drop in pressure to be obtained by this dosage of saline had been secured. Then fluid was allowed to flow from the puncture needle and the pressure was rapidly and markedly reduced.

Considering the relatively small drop of pressure that is obtained by the method of Corbus and his co-workers, is this method as satisfactory as that of lumbar puncture with the drainage of the cerebrospinal fluid? We feel that it certainly is not. The maximum drop that can be expected is infinitely less than is obtained by drainage. Let us recall that, under the system of drainage as suggested by Early and Gilpin (10) and Dercum, (11) after all the cerebrospinal fluid that will flow from the needle has come out and when the manometric reading is zero, it is then possible, by use of suction, to draw out 20 to 40 c.c. more. This will offer a very much greater theoretical possibility of inducing an increase in secretion than can possibly be obtained in man by the saline injections. Thus we feel that spinal drainage by means of lumbar puncture is much more valuable than hypertonic solution injections because of the practical results and also because it causes less discomfort to the patient and does not lead to sclerosis of the veins.

The ingestion of salt has been advocated for the treatment of certain forms of headache, such as migraine. This treatment is based upon the assumption that migraine is caused or accompanied by an increased intracranial pressure and if this increased pressure can be reduced, relief should follow. It has been shown in animal work that the introduction of saline solution into the stomach or into the rectum produces practically the same results and almost as rapidly as when given intravenously. Similar results have been obtained in man. But when given by mouth in capsules or rectally, Foley and Morris find that the salt is usually promptly rejected by the patient. In order to overcome this situation, sodium chloride pills coated with salol, which is not dissolved in the stomach but only in the intestines, have been put on the market.

It seems to us that evidence is still wanting that the pain and discomfort in cases of headache and migraine are the result of increased intracranial pressure or that the treatment as outlined has a therapeutic value. We have not been able to prove that 30 grams of salt, given by mouth in the form of the salol coated pills reduces the cerebrospinal fluid pressure. We have watched the pressure in the spinal fluid manometer for as long as an hour after giving this dose

of salt in this form. In no instance did we find a drop in the cerebrospinal fluid pressure during the period of observation. In fact, in two experiments, there was an increase in the cerebrospinal fluid pressure which we assume was due to the patients' feeling slightly uncomfortable and being a little more rigid than ordinarily.

Clinically, we have tried the effect of the salol coated sodium chloride pills in cases of migraine without any therapeutic benefit. We therefore feel that the value of this form of therapy is still questionable and that if used at all it should be used with an experimental bias on the part of the physician, who should view the method as one with certain therapeutic potentialities and of unproved value.

The work of Weed and McKibben shows that in addition to the lowering of the cerebrospinal fluid pressure produced by the injections of hypertonic solutions, there is an increase in cerebrospinal fluid pressure with the introduction of hypotonic solutions, namely, distilled water. (Chart 2.) Using the same reasonings as for the explanation of the decrease of pressure in hypertonic solution, it is thought that increased pressure with distilled water is due to an attempt on the part of the body to throw fluid out of the blood stream in order to increase the concentration of the blood which has been reduced by the hypotonic solution. In other words, it is assumed that the increased cerebrospinal fluid pressure is the result of fluid being thrown into the fluid system. This may have a modicum of importance in the matter of attempting to reach the cerebrospinal fluid system by medicaments introduced intravenously. Theoretically at least, if one introduces a chemical into the blood stream in strong concentration the tendency seems to be to draw fluid from the central nervous system to the blood stream. On the contrary, if introduced in the form of a hypotonic solution, the tendency should be for an increased secretion of cerebrospinal fluid. Therefore it might seem logical in the treatment of neurosyphilis to use arsphenamine in great dilution in order to facilitate its course into the nervous system. We have been unable to prove experimentally that this makes any difference.

Our method of procedure in this investigation was to introduce 0.3 of a gram of arsphenamine in 200 c.c. of water. In other instances, the same dosage was given in 25 c.c. of water. Watching the effect upon the cerebrospinal fluid pressure as indicated in the manometer, we were unable to note any consistent or definite variation under these circumstances. We are, therefore, unable to say that in the treatment of neurosyphilis by intravenous arsphenamine

that weak solutions are more valuable than concentrated solutions. However, it seems logical and rational, as a result of the effect of hyper- and hypo-tonic solutions to use a weak rather than the strong solution.

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A CASE OF CHRONIC INTERNAL HYDROCEPHALUS DUE TO BLOCKING OF THE AQUEDUCT OF SYLVIVS*

By E. D. FRIEDMAN, M.D.

ASSISTANT ATTENDING PHYSICIAN, NEUROLOGICAL SERVICE, BELLEVUE HOSPITAL;
ADJUNCT NEUROLOGIST, MT. SINAI HOSPITAL, AND LECTURER ON
NEUROPATHOLOGY, NEW YORK UNIVERSITY MEDICAL SCHOOL,
NEW YORK

A certain amount of internal hydrocephalus is nearly always found in tumors of the brain, particularly those of the posterior fossa. But most cases of hydrocephalus have a rather obscure etiology. The milder forms are frequently latent, but may, under the influence of trauma, infection or exposure to sun become more severe and give rise to symptoms. Ependymal proliferation may block either the aqueduct of Sylvius or the foramina of exit from the fourth ventricle and thus give rise to internal hydrocephalus.

The causes of impaired outflow of fluid are tumor, cysticercus and fibrous adhesions resulting from meningitis. There have been a great many theories concerning the genesis of cases in which no definite pathological changes were found. Quincke speaks of a meningitis serosa ventriculorum. Oppenheim mentions the term primary idiopathic hydrocephalus. W. Weber mentions the mechanical causes giving rise to hydrocephalus. (1) Increased formation of fluid in the ventricles. (2) Hindrance to outflow of fluid either through the vena magna Galeni or the foramina of Magendie and Luschka. (3) Diminished resistance of ventricular walls. But none of the theories thus far advanced have adequately explained these cases.

According to Dandy, the absorption of cerebrospinal fluid from the ventricles of the brain is less rapid than its production. The absorption takes place entirely in the subarachnoid space, from which the cerebrospinal fluid passes directly into the blood stream. He divides hydrocephalus into an obstructive type (due to blocking of the exits from the ventricles) and a communicating type in which there is no such obstruction, the hydrocephalus being due to failure of absorption from the subarachnoid space. In his opinion the great

* Presented in abstract before the Section of Neurology and Psychiatry, New York Academy of Medicine.

majority of the cases of hydrocephalus are due to meningitis occurring before or after birth. This is especially true of the communicating type. Bonhoeffer considers a mild increase of the cells in the

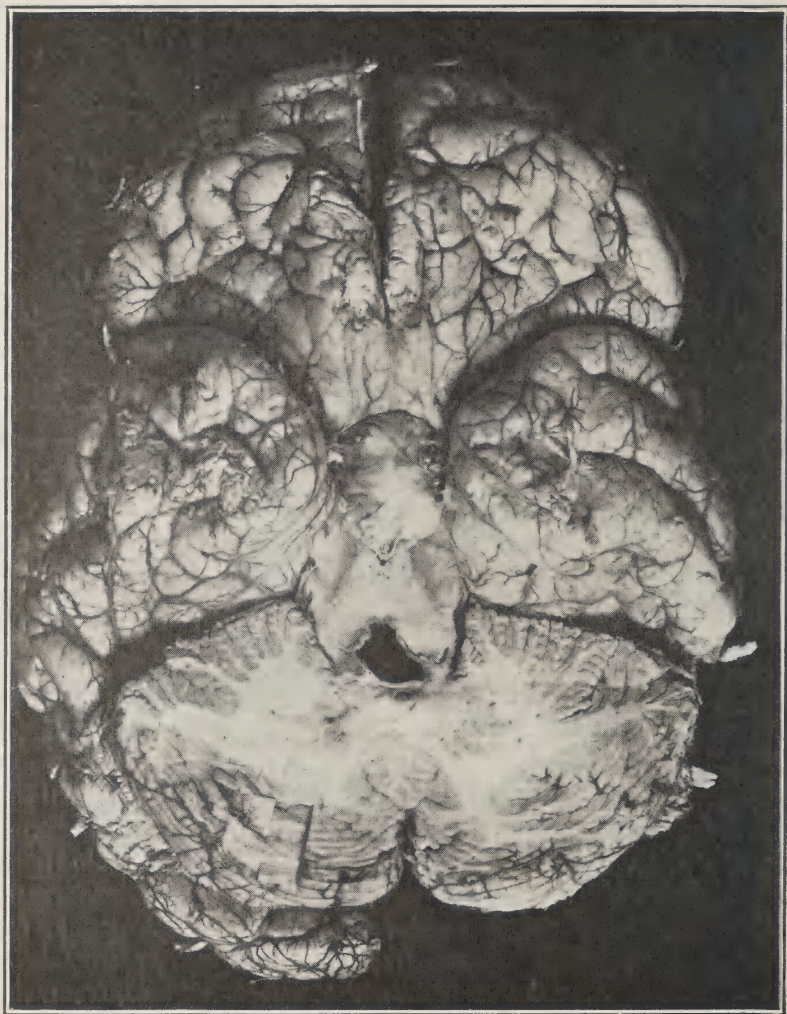


FIG. 1. Basal surface of the brain, showing the cyst, occupying the entire interpeduncular space. There is no trace of normal structures in the floor of the third ventricle.

cerebrospinal fluid a usual phenomenon in acquired hydrocephalus.

Magendie (1) described occlusion of the aqueduct of Sylvius in 1842. Bourneville and Noir (2) reported a total occlusion of the iter. Touche (3) was the first to attribute the hydrocephalus which

he described to blocking of the aqueduct. Spiller (4) cites two cases in one of which the aqueduct of Sylvius was not patent. He studied the case microscopically and found hypertrophy of the glia. Guthrie (5) described studies in hydrocephalus in patients who died of meningitis. He emphasized the relationship between these two conditions and included eight cases of blocking of the iter in his report. Schlapp and Gere (6) have reported a number of cases of congenital obliteration of the aqueduct. All their cases were children. Dandy and Blackfan (7) reported four cases of obliteration of this canal and later Dandy (8) reported two additional cases of blocking of the aqueduct.

The symptoms of internal hydrocephalus are very varied and the clinical picture may be very confusing. The hydrops of the ventricles may give rise to the syndrome of an intracranial lesion with or without focal signs. The symptoms include muscular rigidity, transitory clouding of consciousness, visual disturbances and often bitemporal hemianopsia from pressure of the dilated third ventricle on the hypophysis. The knee jerks are frequently absent, due to hydrorrhachia. The process may become arrested and leave blindness in its wake.

The symptoms of brain tumor are frequently simulated, especially those of the cerebellum. There are often weakness of the legs and tremulousness. Patients also frequently complain of increase in headache, when the head is suddenly thrown backward. Of the cranial nerves, the sixth is most frequently involved, because of its well known vulnerability. Occasionally exophthalmos is found. Oppenheim reported a case with involvement of the labyrinth leading to unilateral cerebellar signs. Acute exacerbations of the clinical signs are frequent. The symptoms may be so vague (headache and dizziness) as to resemble hysteria.

In the child, the separation of the sutures of the skull helps to make the diagnosis. In the differential diagnosis from tumor of the brain, the following considerations, according to Oppenheim, are of value: The congenital origin of hydrocephalus is frequently demonstrated by the size and shape of the head. There is usually a history of a long course with remissions often of years duration. The absence of definite focal signs in hydrocephalus is extremely important. The findings which suggest hydrocephalus are the development of symptoms following trauma or infection and a course which is unusual for brain tumor. Cerebellar ataxia, asynergia and stiffness of the neck are also frequently observed. Spinal puncture may help in the diagnosis. It usually shows lessening of the fluid pressure. The Queckenstedt maneuver (compression of the jugular veins) might be of service in this connection. In some cases of hydro-

cephalus, rhinorrhea may supervene (autodecompression). Choked disc is much more common in the acquired form of hydrocephalus.

We have recently had occasion to observe a patient with marked hydrocephalus, as a result of blocking of the aqueduct of Sylvius. The anatomical findings were very similar to those described by Schlapp and Gere.

Anna A. Age twenty-two. Single. Admitted to the service of Dr. B. Sachs at Mount Sinai Hospital, January 8, 1922; died January 10, 1922.

Family History: Negative.

Previous History: No instrumental delivery, developed normally up to the age of two. Then patient had an attack of "brain fever" with coma and paralysis, more marked on the left side. She was acutely ill for two weeks and after this attack, stopped walking and could not talk. Her illness continued for three years. She then improved somewhat. At eight years she had typhoid fever and is said to have had convulsions at that time. She went to public school, graduating at sixteen. Her periods began at thirteen. They were regular but within the last year they have been long delayed. At one time there were no menses for eight months. During the last three months they have again been regular. Patient was injured in a pogrom in Russia in 1905.

Present Illness: For three years patient has had frequent headache and pain in the back of the neck. She also suffered from dizziness and pain in the eyes. There was no vomiting or scotoma. For the last year the headache has been growing steadily worse.

Physical Examination: Head was somewhat large. There was facial hirsutes (of male type). Pupils reacted well to light and accommodation. The isthmus of the thyroid was palpable. The breasts were well developed. There was a systolic murmur at the pulmonary area. The abdominal wall was rather obese. The patient showed an acne-like eruption. There was a congenital deformity of the fourth toe of both feet. Patient's mentality was distinctly retarded. In standing, she swayed a little and maintained the erect posture with difficulty. There was a positive Romberg sign. Both discs were pale, the left more so. There were slight nystagmoid twitches. There was a questionable weakness of the external recti, more marked on the right. There was slight weakness of the left face. The innervation of the palate was poor. The visual fields showed some contraction. A coarse tremor was present in both hands and a fine tremor of the tongue was noted. There was distinct ataxia, more marked in the lower extremities; adiadochokinesis was observed, more on the left. Both lower extremities were weak, the left more so. The abdominals were present though not overactive. The deep reflexes were generally exaggerated. Babinski's sign was positive on both sides. There was a tendency to bilateral pes cavus. There were no sensory changes. The gait was spastic ataxic. The white cell count was 14,000. The differential count showed poly-

nuclears 85 per cent, lymphocytes 15 per cent. The temperature varied from 98 to 99.8, pulse from 96 to 120, and respiration from 20 to 24. The blood pressure was 140/90. The urine was negative. The blood Wassermann was negative.

On the second day after her admission to the hospital the patient while on a bed-pan, expelling an enema, fell out of bed in a faint.

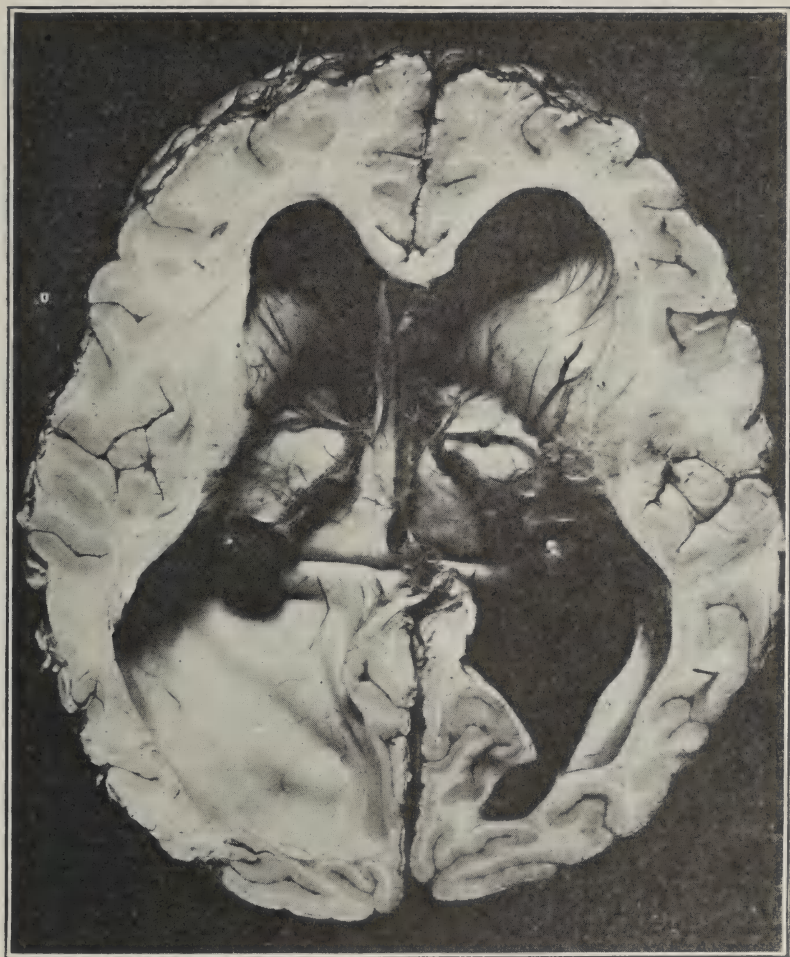


FIG. 2. Transverse sections showing enormous dilatation of the lateral ventricles.

Respiration stopped and the heart continued to beat for a few minutes, but in spite of active stimulation, she died promptly. The clinical diagnosis varied from atypical multiple sclerosis with chronic ependymitis to cerebellar neoplasm with secondary symptoms. There were distinct pituitary stigmata (abnormal hair distribution, amenorrhea and a tendency to obesity). The patient was not in the hospital

long enough to permit an x-ray examination of the skull or lumbar puncture.

Anatomical Findings: Brain and Meninges. The dura was markedly thinned indicating increased intracranial pressure of long standing. In places it was reduced to the thinness and translucency of tissue paper. The pia-arachnoid on the dorsolateral surface was

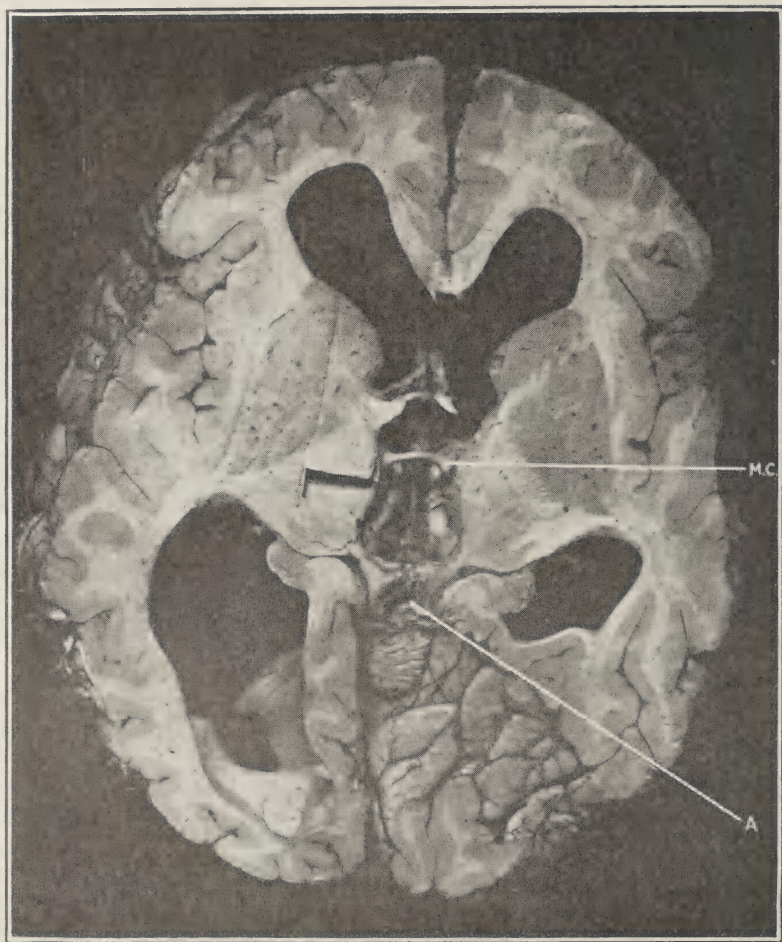


FIG. 3. Transverse section showing dilatation of third ventricles and the blind sac (a) interrupting the aqueduct of Sylvius. The middle commissure is shown here markedly stretched and thinned.

smooth and glistening and showed no abnormal thickening. The gyri were definitely flattened. The brain substance on palpation showed marked decrease in consistency. On raising the orbital lobes of the brain, a large cyst located in the interpeduncular space and occupying the large, eroded sella turcica, was found. In attempting to expose the cyst more fully, it was punctured and crystal-clear fluid escaped under pressure leading to collapse of the cyst. Sagging of the cerebral

hemispheres followed the escape of the fluid. The cyst gave the appearance of being connected with the infundibular duct of the hypophysis, but further observation showed it to be an extensive hernia in the floor of the third ventricle (Fig. 1). The pituitary body was somewhat compressed; otherwise showed no changes. The meninges at the base showed no thickening. The structures in the interpeduncular space were almost completely replaced by thin membranes, which formed the inferior surface of the above described cyst. No trace of mammillary bodies. The tuber cinereum could not be identified in the wall of the cyst and the optic chiasm was



FIG. 4. Section showing part of the mass (G) imbedded in floor of the fourth ventricle. Anteriorly it completed the aqueduct of Sylvius.

markedly thinned and adherent to the cyst. The pons and the medulla also showed evidence of compression. On sectioning the brain, the lateral ventricles (Fig. 2), the third ventricle and anterior portion of the aqueduct of Sylvius (Fig. 3) were found markedly dilated. The aqueduct of Sylvius was interrupted posteriorly by thickened membrane cutting off its connection with the fourth ventricle. Posterior to this membrane a small solid mass was found in the floor of the fourth ventricle, directly behind the opening of the aqueduct of Sylvius into the fourth ventricle, obstructing the communication between them.

Microscopic Anatomy: Section from the small mass showed it to consist of proliferated neuroglia with many small, somewhat distorted

ependymal cysts. In some of these cysts, the lining cells were exfoliated.

The post-mortem findings were a surprise. The combination of pyramidal tract and cerebellar signs was indeed suggestive of multiple sclerosis and since chronic ependymitis often forms part of the anatomic picture in this disease, it was felt that that was the most likely clinical diagnosis. Cerebellar neoplasm was thought of but in the absence of choked disc, was deemed improbable.

There was no evidence of increased intracranial pressure aside from the headache. There was no slowing of the pulse, no vomiting. This may have been due to the long duration of the patient's illness, with its onset in infancy and the consequent slow accumulation of fluid.

The pyramidal tract signs of course are easily explained by the hydrocephalus. Cerebellar manifestations, too, are common in this condition. The pituitary disturbances found their explanation in a marked herniation of the floor of the third ventricle with consequent pressure on the hypophysis.

The sudden death may have been due to extreme stretching of the tuber cinereum. Bailey and Bremer (9) recently pointed out in their experimental work that sudden death commonly followed puncture of this region of the brain. It was also noted that the patient had a somewhat Parkinsonian attitude. This was probably due to pressure of the internal hydrocephalus on the basal ganglia.

I wish to express my indebtedness to Dr. B. Sachs for permission to report the case and to Dr. J. H. Globus of the Department of Neuropathology for helpful suggestions.

74 East Ninety-first Street.

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SOCIETY PROCEEDINGS

NEW YORK NEUROLOGICAL SOCIETY

THE FOUR HUNDRED AND NINTH REGULAR MEETING, JANUARY 8,
1924, WITH THE ACADEMY SECTION OF NEUROLOGY AND
PSYCHIATRY. DRs. E. G. ZABRISKIE AND
H. A. RILEY PRESIDING.

The following program was carried out:

A CASE OF PAGET'S DISEASE OF THE SKULL, ASSUMING A MALIGNANT CHARACTER

Dr. Foster Kennedy said: The patient, a married woman, forty-four years old, does not show the usual characteristics of Paget's disease, such as deformities of the long bones, and bowing, but since 1918 she has not been well, she has vomited, felt dizzy, seen double, has had frontal and occipital headaches, and has staggered when walking. Physical examination shows a definite deformity in the left frontal area, a large boss in the left frontal region of the skull, perhaps a meningioma with periostitis and exostosis. There is a well-defined area of bulging. The bones of the face are normal. There is an area of tenderness over the exostosis back to the occipital bone, with a definite, spongy, edematous sensation to touch. There is papilledema of both fundi, of a chronic type, but not much tortuosity of the vessels, no exudate. An opinion was advanced that she had an affection of the right face, but I do not note this. She has no abdominal reflexes on the left side, and quite a definite Babinski on the right. She has a general motor incoördination. The Wassermann is negative both in blood and spinal fluid; globulin is 2+. Blood chemistry is normal, urine normal. Her husband is well and she has had four normal children. It has been claimed that Paget's disease is an evidence of tertiary lues, but I do not find any sign of that here. The X-ray is entirely typical of Paget's disease, showing the typical tufty appearance. The patient was treated by deep X-ray therapy, and experienced a severe reaction after the treatment, consisting of vomiting, dizziness, and staggering. This was ameliorated by lumbar puncture. She has improved materially under treatment. I do not believe this growth is becoming malignant. Paget's disease rarely has intracranial complications, but it is not unknown. In this case we have resisted the temptation to secure a specimen.

Discussion: Dr. Leon T. LeWald (by invitation) said: In view of 35 cases of Paget's disease studied radiographically, I can review some of the interesting points that Dr. Kennedy brought out, especially that in this case there may not be any malignant change. The lantern slides I show to-night will illustrate this. Paget reported 12 cases in 1876. A number of his cases are said to have had sarcomatous change, but I would wish to question the nature of the so-called sarcomatous changes, as in some cases which I have seen the specimen taken from the facial bones which were greatly involved showed only osteitis or cystic degeneration on microscopic examination. We have seen 35 cases in twenty years. Many early cases of Paget's disease are not recognized. In one case there was cystic degeneration of the inferior maxillary bone. Dr. Abbe was much interested and thought of making a complete resection of the lower jaw as he thought it was sarcoma until the X-ray revealed the limited character of the lesion. There was no definite tumor formation and the specimen showed it was cystic degeneration. The man is alive and well today, about eight years later. The second case is of involvement of the superior-maxillary bone. Dr. Abbe removed a section from this case of Paget's disease and found proliferation of bone, but no malignant change. Paget is said to have found that fractures in these cases do not unite, but we have not found this to be true. Cases are sometimes mistaken for osteomyelitis and operated on for that condition. As far as sarcomatous degeneration is concerned, this must be regarded as a rare coincidence. The point of etiology is very important. In 35 cases we have found no evidence of syphilis. In one case a four plus Wassermann was reported, but on careful inquiry it was found to be a report for the man in the next bed.

Dr. J. Ramsay Hunt said: Is there any possibility that this could be a condition like dural tumor, upon which Cushing has laid such stress? That also develops a boss, but it has a different appearance from that of Paget's disease of the skull.

Dr. Foster Kennedy said: In answer to Dr. Hunt, I would say that we considered very carefully the possibility of this woman having an endothelioma, with hyperostosis over it. We abandoned that idea because in dural tumor, with exostosis there is a hard, ivory-like structure, which is like a billiard ball. There is no pain over it. Here the X-ray showed irregular thickening of the bone. It was new bone formation with absorption going on, which is typical of Paget's disease, the latter being a sub-acute inflammatory process, rather than primary bone hardening. The lesions of the tables of the skull in dural endothelioma are hard, whereas in Paget's disease the consistency is characteristic. It is rare to have symptoms of intracranial pressure, and we have to consider the differential diagnosis very carefully. We had to compromise and extend the picture and symptoms to perhaps inflammation of the skull itself, which would be sufficient to account for the symptoms seen.

I. PRIMARY OPTIC ATROPHY AS A LOCALIZING SIGN
IN FRONTAL LOBE TUMORS

(ILLUSTRATIVE CASES)

Drs. E. D. Friedman and J. H. Globus said that the significance of retrobulbar neuritis with development of a central scotoma in the localization of infrafrontal tumors, although well known, had been stressed by one of our presidents, Dr. Kennedy. We have had occasion to observe two cases illustrating this phenomenon, in both of which we were able to substantiate the clinical diagnosis by the autopsy findings.

Case 1. Hospital No. 218792. Male, born in Russia. Aged forty. Tailor, married. Admitted to the service of Dr. B. Sachs at Mt. Sinai Hospital April 17, 1922, died May 26, 1922. *Chief Complaint:* Dizzy spells for three years. *Family History:* Negative. *Previous History:* The history was obtained with difficulty. Patient responds slowly and cannot concentrate. His illness began with dizzy spells three years ago. He would fall to the ground and lose consciousness. The left side of the body would stiffen out and there was involuntary micturition. These attacks would come on once or twice a week. Only occasionally did he experience headache. At the same time there was noted facial weakness on the left side. He also showed mental changes with delusional content; became irritable and quarrelsome, neglected his business and showed an alteration in personality. More recently his memory began to fail and he would frequently miss his station on the way home.

Physical Examination: The general medical examination proved negative. The patient exhibited emotional instability; at times he was euphoric, at others, quarrelsome. He was disorientated, facetious. He showed perseveration, yawning and slow cerebration. Speech was hesitant, thick and explosive. The sense of smell seemed impaired. The pupils were sluggish to light. There was a left hemiparesis involving the face also. He exhibited spasm in the left orbicularis palpebrarum. The left corneal reflex was diminished. The knee jerks and ankle jerks were active, the left more so. Babinski sign was present on the left, abdominals were diminished, especially on the left. Percussion tenderness was present over the right frontal area. There was marked incontinence of urine. Lumbar puncture revealed clear fluid, under increased pressure and containing 24 cells to cmm. Spinal and blood Wassermann were negative. Blood pressure was 145/110. Urine was negative. Temperature, pulse and respiration were normal. *Fundi:* O.D. Obscure disc outline with suspicious elevation of the upper third of the disc (less than L.D.). Temporal half is pale and flat looking. O.S. same as O.D. except less marked. *Diagnosis:* Bilateral low grade primary optic atrophy with suspicion of early superimposed papilledema. Due to the patient's condition, we could not demonstrate scotomata. The patient became more stuporous, and rapidly got worse. Ventriculography was done as a preliminary to operation but the patient died soon afterward.

Post-Mortem Findings: After reflecting the dura, the brain showed evidence of increased intracranial pressure; the gyri were flattened, the sulci, shallow, the cortex pale. The frontal poles were adherent to the dura. The dura was in its turn adherent on its under surface to the orbital plates of the frontal bone and the cribriform plate of the ethmoid. Upon removing the brain, a tumor involving the dura, in the mid-line between the inferior surfaces of the frontal lobes was seen. The tumor was circular in shape and about two inches in diameter. The olfactory tracts on both sides were imbedded in the mass. The tumor was reddish, soft and invaded the brain substance of the right frontal lobe. It could be readily separated from the left frontal lobe into which it had imbedded itself. The brain showed no other pathological changes. Section of the brain showed that the large tumor was disc shaped, flattened in its vertical diameter and was shelled out, with ease, from the brain tissue in which it lay. It had penetrated about one and a half inches symmetrically into both frontal lobes. It did not encroach upon the ventricles or the corpus callosum. Small recent subependymal hemorrhages were found in the left lateral ventricle. The ventricles were small but not distorted. On microscopic examination the tumor mass proved to be an endothelioma. ?

Case 2. Hospital No. 219785. Male, borne in Austria. Age forty-eight. Iron worker, married. Admitted to the service of Dr. B. Sachs at Mt. Sinai Hospital, May 24, 1922, discharged July 24, 1922. *Chief Complaint:* Lethargy for two months. *Family History:* Wife had one miscarriage at seven months. *Previous History:* In the United States nineteen years. Never seriously ill. Head was struck in the left frontal region by a brick four years ago. There was no fracture, just suture. *Present Illness:* In May, 1921, patient developed a serious attack of headache without vomiting. He seemed, however, tolerably well until April, 1922, when his wife noticed that he "mixed up words" and on May 20, 1922, he went to work but did not return home on time. He was supposed to have been struck on the head. There is no evidence to support this statement. On his return home, he could not talk and had a violent headache. Recently he showed a tendency to drowsiness, became irritable and his memory became poor. His vision also began to fail. His hearing was impaired on the left. There was difficulty in urination but no vomiting.

Physical Examination: Sense of smell was impaired on the left. There was left exophthalmos. Vision in the right eye was better than in the left. The visual field on the right was contracted. On the left there seemed to be an enlargement of the blind spot. The pupils were sluggish to light; the right was larger. There was mild external strabismus, due to weakness of the left internal rectus. There was slight left ptosis. Right facial weakness was evident. The right corneal reflex was diminished. The knee jerks were elicited with difficulty. Abdominals were decreased especially on the right.

The right plantar response was not definitely flexor in type. The patient carried out commands but sometimes had difficulty in recognizing objects named by the examiner. Sometimes he could not name objects on seeing them or when they were placed in his hand, but could describe their use. Read and understood what he read. There was no apraxia. At times he missed a letter in writing or substituted one letter for another. This applied also to figures. The difficulty seemed to be one of word naming and word finding. *Fundi*: O.D. disc outline is blurred. Surface capillaries injected. Suspicion of elevation. Arteries tortuous. Early papilledema. O.S. disc outline clear but papilla is pale. There was slight percussion tenderness of the skull on the left. Blood pressure was 100/70. Hearing was normal. Vestibular apparatus was normal. Urine was negative. Blood and cerebrospinal fluid Wassermann were negative. Fluid was contaminated with blood. X-ray examination of the skull was negative. Exploration of the left frontal area was performed but no tumor was found. The patient left the hospital but returned on July 23, 1923.

The physical examination now showed some changes in the status. There was weakness of the entire right side, more marked aphasia, of mixed type, alexia and agraphia, akinesis and general mental deterioration. He was euphoric, indifferent to his surroundings, and exhibited inadequate emotional responses. He urinated on the floor during the examination. He was undisturbed by the fact that he could not express himself and frequently exhibited perseveration. *Ventriculography* showed an obliteration of the descending horn of the left lateral ventricles. This led to temporoparietal exploration with negative results. Patient died several days later.

Post Mortem Findings: The meninges were normal. There was evidence of increased intracranial pressure; the gyri were flattened and the sulci, shallow. Beneath the left frontal lobe and extending into the markedly widened Sylvian fissure there was an encapsulated tumor about 7 cms. in diameter. This tumor was readily shelled out of the brain tissue. The left lateral ventricle appeared to be displaced to the right side, and was compressed; the right lateral ventricle was dilated. The cranial nerves aside from the first two, presented no abnormalities. The left side of the sella turcica was eroded and partly destroyed. Microscopically the tumor proved to be an endothelioma.

In the first case, due to the presence of bilateral primary optic atrophy, with diminution of the abdominal wall reflexes, the diagnosis of multiple sclerosis suggested itself at first but was rapidly abandoned because of the features pointing to a right frontal neoplasm. The optic nerves were not directly involved in the tumor mass as found at post mortem but there was displacement of brain substance and herniation downward, sufficient to make traction on the optic nerves and thus produce a result similar to that of actual compression.

In the second case, because of the failure to find a tumor in the left frontal region at the first operation, and because of the subse-

quent ventriculographic findings, a temporoparietal exploration was carried out in the face of manifest clinical evidence of left frontal lobe lesion. The autopsy findings, as already shown, proved the correctness of the first clinical impression of the case and again emphasizes the significance of ipsilateral primary optic atrophy as a localizing sign of great value in infra-frontal tumors.

Discussion: Dr. Philip Goodhart said: I would like to ask the readers of the paper whether the optic atrophy involved both of the discs; or was the condition that of a low grade optic neuritis on the side opposite the lesion, with a primary atrophy on the side of the brain affected? We recall the latter condition as described by Foster Kennedy some years ago as characteristic of expanding lesions of the frontal lobe. If in the cases presented there was, from the very beginning, atrophy rather than neuritis on both sides, this symptom as one suggestive of frontal lobe lesions becomes very important. It would seem, however, that for a primary atrophy in this location the lesion in the frontal lobe must be mesially located and the character of the growth, in other words its consistence, its pathological type, would have a bearing in the production of primary optic atrophy.

Dr. Foster Kennedy said: Ventriculography, as a method of exploring the ventricle, is not superior to looking at the eye grounds and drawing deductions therefrom. We must not sacrifice essential observations for those of second grade. When we have a piece of nervous tissue, the function of which we know and can estimate, and we find that this function is gone, it is right to say that the lesion is in the structure subserving that function. Therefore diagnosis should have been made on that fact, rather than by bedevilment with the ventriculogram. I am perhaps prejudiced, but for twelve years I have been finding cases with atrophy on one side and papilledema on the other, and that is definite and precise information. Nothing but pressure on the optic nerve can produce central scotoma and primary degeneration of the masclar bundle. If the tumor is in the middle of the frontal lobe there will be papilledema on each side to begin with, but as soon as the tumor grows down and strikes the optic nerve, central scotoma will be evident, and primary optic atrophy will be superimposed on the papilledema.

Dr. E. D. Friedman said: We might effect a compromise and say that both methods are valuable. Dr. Kennedy insists on the clinical side of the cases. I endeavored to get a diagnosis from clinical evidence, and in Case 2, in spite of the fact that the tumor was not found at operation, we thought that the lesion was frontal. If the technic of ventriculography had been properly carried out it would have shown that the ventricle was blocked because the tumor invaded the Sylvian fissure. Proper technic would have proved pressure on that. In Case 2 there was right-sided papilledema and left-sided optic atrophy.

Dr. Globus said: An exploratory operation was carried out in the second case with the diagnosis of frontal neoplasm having been made

on clinical grounds only. The tumor was not found and only then an attempt was made to use other diagnostic methods. The ventriculography led us away and astray from the original diagnosis.

PSYCHOGALVANIC REFLEX AND ITS CLINICAL APPLICATION

Mr. David Wechsler (by invitation) gave this paper: The psychogalvanic reflex is a term that has been applied to those electrical variations in the conductivity of the skin which may be provoked by certain psychic stimuli and in particular by the emotions. The psychic stimuli which have been reported as capable of provoking to galvanic response are very numerous. They include: any strong sensory stimulus, as the sound of a gong, the prick of a pin, etc.; recollection of a pleasant or unpleasant experience; the association experiment; mental calculation; changes in the state of attention; and many others, ranging anywhere from the winking of the eye to subconscious suggestions. In spite of their diversity, however, the consensus of opinion of investigators is that a stimulus is effective in provoking a galvanic response only in so far as it is capable of arousing an effectively toned psychic process, either conscious or subconscious.

Skin alone is the organ involved in the mediation of the "reflex," which is also localized to those portions of the skin that are provided with sweat glands. The "reflex" is due to the diminution of the counter-electromotive force of polarization set up by the polarizing current, this diminution being effected by the added secretion of sweat that is provoked each time the individual in the circuit responds affectively to a stimulus. The galvanic response is unobtainable when the source employed is an alternating current of high frequency, namely, one that does not polarize the tissues as it traverses them (Prideaux).

The practical application of a galvanic response will naturally depend upon the nature of the other phenomena with which it correlates, its reliability as an index of those phenomena, and the ease with which the method may be applied. Clinically, the results obtained have shown themselves significant in at least five different fields.

The first application of the "reflex" has been in connection with the association experiment which has been employed as a means of detecting so-called complex indicators, chiefly by the comparative study of reaction-times of various stimulus words, those with relatively long reaction-times being considered as the complex indicators. A number of investigators have made experiments to compare the galvanic response with the reaction-times (Petersen and Jung, Veraguth, Binswanger, and Smith), and all have found that the P.G.R. is by far the more sensitive of the two indicators. The P.G.R. is not only more easily elicited but it gives indication of positively as well as negatively toned affective memory.

The second application has been to the study of the degree of affective disturbance in certain mental disorders. The results in

these experiments have shown remarkable correspondence between the change of affectivity in the subjects as revealed by clinical response. The galvanic reaction of subjects with mental disorders which show the least affective deterioration have been found to approach, as regards both magnitude of galvanometric deflection elicited and allure of the galvanometric curve, the response manifested by a normal individual. This, for instance, is the case in the anxiety neuroses. On the other hand, in those psychoses where there is a great deterioration of affectivity the individuals deviate most from the normal. Such, for instance, is the case in dementia precox and in the late stages of paresis. (Prideaux, Gregor and Gorn). Gregor and Gorn have furthermore found the method useful to differentiate forms of true catatonia, patients which usually gave no galvanic response at all or only insignificant ones; from melancholia patients who frequently gave spontaneous galvanic reactions on the basis of the nature of their psychogalvanic reactions. The emotional reactivity of the hysteric is below normal, and shows marked deterioration.

The third application of the P.G.R. has been to distinguish between true and hysterical anesthesia and analgesia. The hysteric is an individual who not only pretends (outwardly) to experience what he does not feel, but also frequently disclaims the feeling of what he really does experience (though not of course consciously). Now, experiment has shown (Veraguth) that when appropriate stimuli are applied to the region of the skin over which the hysteric claims to have lost sensations, the patient responds with galvanometric deflections to the stimulation of those pretended anesthetic and analgesic regions. On the contrary, patients suffering organic analgesias or anesthetics (*e.g.*, tabes or syringomyelia) give no reactions under corresponding stimulation.

Finally, Godefroy, by making some modifications in the technic of measuring the galvanic response has been able to record the continuous slight changes of affectivity agitations, which characterize certain phases of exophthalmic goiter. He has, in fact, shown that the curves obtained are markedly different and distinguishable from those of healthy individuals, and further, that as the patients improve, their curves approach more and more those obtained from normals.

My own researches have for the most part been devoted to the problem of the possibility of employing the P.G.R. as a means of measuring the affective responsiveness of individuals so that one person's reactions might be compared quantitatively with that of any other. For this it was necessary to study the electro-physiological conditions which might influence the magnitude of the response and to discover improved means of technic which would eliminate practical difficulties. As illustration of the first, it might be mentioned that the magnitude of the reflex varies with the time of the day, the region of the body to which the electrodes are attached, state of fatigue of the subject, and most of all upon the intensity of the current passing through the subject's body. In connection with the ques-

tion of technic the problem was first to devise a circuit by which one could conveniently control the intensity of the current passing through the body, and, secondly, of constructing an apparatus of small size which would enable the experimenter to record photographically the galvanometric deflections without necessitating the introduction of the subject into a dark room or distracting him and frightening him by the sight of the measuring instruments. Both of these, I believe, I have been able to solve successfully. My apparatus is described in another article.*

Discussion: Prof. S. R. Woodworth (by invitation) said: I would like to emphasize the fact that we have here some definite phenomena, which we would not expect to find related to emotional response, but which, upon analysis, we find bear a real relationship to the emotional reactions of individuals. As I understand it, the electrical response occurs with the change in emotion and is more adaptable to sudden emotion than to a long-continued state of mind. The conditions obtained involve the element of surprise or suddenness, which causes the individual to readjust himself to the situation, rather than requiring him to go through a steady mental performance. I do not know whether one could expect this phenomena to give a general measure of emotivity that would be a companion piece to degree of intelligence. I do not know whether we can speak of one person as being more emotional than another in the general sense. We can perhaps speak of emotivity of certain kinds. One person is more subject to fright, another to lust. Emotions of a certain kind may arise in one person and not in another, so that I do not feel that we have here anything comparable to an intelligence test. I do feel, however, that the definite response obtained is worthy of attention, and I would like to hear this discussed more fully.

Dr. Foster Kennedy said: Fifteen years ago when I was working on this instrument I developed a prejudice against it. In testing for pleasurable ideas I had a chocolate cream put in my mouth in the dark, and derived very slight pleasure from it. I do not think that much progress has been made with this instrument. The present knowledge seems to be in the same state as our former knowledge on the subject. If this instrument has been worth pushing there has been considerable energy spent on it, but what is the good of reducing to figures things that we can perfectly well express in ideas. It seems to be a waste of time. Mr. Wechsler's work would show that he thinks we suppose an individual suffering from conversion hysteria is highly emotional, but we do not think so. That person is really underemotional, so that, if he finds in conversion hysteria a drop in emotivity from 90 to 36, that is simply a corroboration of common clinical experience.

Dr. Oberndorf said: When repeating Jung's experiments, I did

* Wechsler, D. Sur le Technique du Réflexe Psychogalvanique. (Demonstration d'un Nouvel Dispositif.) Compt. Rend. des Séances de la Soc. de Biol. Séance du 3 Dec. 1921. T. LXXXV.

some work on association tests, and I wondered whether clinical experience does corroborate these tests. The normal person's response shows very little retardation because his complexes are under control. As Dr. Kennedy says, in insane states, there are more deflections of the galvanometer than normal, and very little energy attaches to stimuli. I doubt whether this gives a true measure. In dementia precox emotion may be felt but not exhibited. The difficulty in these cases is not finding of complexes, but to know how to deal with them after we get them. The real question is how to remedy the complex.

Dr. Goodhart said: Mr. Wechsler has brought out an interesting fact in his experimental investigation, namely, that in the case of the so-called hysterical patient there was a definite deflection of the galvanometer. Here we have evidence of what is generally believed, namely that there is a difference between the conscious emotional reactions of the hysterics to a painful stimulus and that of the unconscious. The painful stimulus is evidently recognized as such, is, so to speak, automatically differentiated though the element of recognition in the conscious is suppressed. The galvanometer therefore furnishes an instrument which would seem to be an aid in study of the psychology of this interesting field of clinical work.

Dr. Joseph Smith said: Mr. Wechsler has classified idiots, as regards general emotivity, on a higher plane than persons suffering with general paresis. We know that in the early stages of general paresis there is a great increase in emotivity.

Dr. Rosett said: I looked into the subject some time ago. The assumption that the galvanometric changes are due to emotional states does not agree with a number of facts. One of these facts is that a continuance of a given emotional state does not result in a correspondingly continued galvanometric response. It appeared to me indeed that the galvanometer registered changes in the state of attention rather than affective states. When, for instance, a strong galvanometric response is obtained immediately the subject hears the word "lamp-post," it is rather difficult to conclude that the emotion produced was strong enough to initiate the activity of the sweat glands to which the galvanometric response is assumed to be due. On the other hand, any changed state of attention that might have resulted from hearing the word "lamp-post" might result immediately in a change of posture, implying a widespread rearrangement of the pattern of muscular contractions. Such muscular activity results in action currents and would be registered by a movement of the galvanometer. The disproportionate degree of response in such cases may be due to causes which so far remain undiscovered.

Mr. Wechsler (closing) said: Since the time of Peterson and Jung a great amount of progress has been made. This is not a classification of mental disorders in regard to affectivity. The figures of the psychogalvanic reflex correspond closely to ideas already made. Intelligence tests are used to get the status of normal people. We do not apply them to idiots. We want to differentiate fine distinctions between people of average capacity, and those above the average.

Peterson and Jung applied these tests without means to control electromotive force. We have proved this is not due to muscular contraction because intensity of current by muscular action is too low in order, such as millivolts. If you have 5 to 10 volts, it is not muscular response. There is also a latent time between response and stimulus, equal to that required for activation of the sweat glands. It does not require new secretion to free electromotive force. The polarization that takes place can be controlled. By switches we can change the galvanometer into an amperemeter, controlling the amount of current passed into the human body. Both small and large changes in affect can be measured. We referred to consciousness and unconsciousness of the galvanic reflex. There have been experiments on soldiers suffering from parietal injuries with partial loss of sensation. They did not feel but responded to stimuli below the center of consciousness. This method can be used for differentiating lesions of the nervous system associated with anesthesia. There is a difference in types of dementia. Hebephrenic types respond in a manner approximating normal. They are different from the catatonic type.

SOME OF THE FUNDAMENTAL PROBLEMS OF PSYCHIATRY

(Author's Abstract)

Dr. Stewart Paton said: One of the fundamental problems of psychiatry relates to the meaning and application of the word "psychiatry." Are we justified in using a word coined by physicians when they were engaged to a large extent in describing the symptoms "to correspond with certain ideal forms of disease"? The word "psychiatry" suggests the study of specific types of disease, and does not usually include the broader biological conception of disorders of adjustment. The use of a term with specific limitations has theoretical as well as practical limitations and theoretical as well as practical disadvantages. In order to understand some of the fundamental problems of psychiatry it is necessary to study mind in the process of organization. This period of organization corresponds with the life of the embryo. Although libraries are filled with literature bearing on the ancestral and post-natal periods, there is very little information available about the nature of the processes active during the period of embryonic development. The primitive sensory and motor responses developed during embryonic life are very important factors in determining the subsequent organization of the higher mental processes. If we knew more about the nature of these processes and the conditions under which they operate, we should be in a better position to understand the supplementary forms of conscious adaptation that are developed in order to enable the individual to adjust to a wider and more complicated environment. There exists during the life of the embryo an excellent opportunity to investigate the relation of muscular tonus to movement and also the relation of the various movements in preparing the way for the higher forms of conscious adaptation.

The various experiences registered before birth undoubtedly have

a large share in determining the organization of the mysterious field of the unconscious. These experiences, although they may be directly represented in the field of consciousness, may induce modifications in the various forms of conscious adaptation. It is also possible during the life of the embryo to study the primitive forms of organic memory which include the phenomena of registration, retention, and the capacity to recall impressions.

Information already obtained from the study of the reactions of the embryo have thrown light upon the nature of such processes, as inhibition, and repression, that are of fundamental importance in the study of the mind.

What is particularly needed at present is more active and intelligent coöperation between groups of workers who are attacking the problem of mental organization from many different standpoints.

Discussion: Dr. Stragnell said: I agree with Dr. Paton that by paying attention to the problems of embryological psychology we should be able to get some light on later stages of development. I do not follow him, however, when he says that the psychiatrist is on the defensive in regard to terms of common usage, although there are certain workers who are opposed to accepting the findings attached to extremes of differentiation, but they are not at all reluctant to follow the lines suggested by Dr. Paton.

Dr. Rosett said: Inhibition generally means inhibition of muscular contraction, at least to the neurologist. I do not know what it means to the psychiatrist.

Dr. Paton (closing): I think I am with you as regards the meaning of inhibition. In regard to the meaning of terms, I do not think there is any disagreement. We are often carried away by the use of terms and no two psychiatrists will agree as to the meaning of the word "complex." To some it means repression, but what is repression from the neurological standpoint? One stronger impulse may cut out an impulse that is not quite so strong, or an impulse may die out from diminution of the neural potency. Again, when we use such terms as "unconscious" and "subconscious" we introduce elements upon which we are not informed. The meaning changes as our information becomes greater.

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

REGULAR MEETING, JANUARY 15, 1924, F. K. HALLOCK, M.D.,
PRESIDENT, IN THE CHAIR

CONTRIBUTION TO THE STUDY OF APHASIA AND APRAXIA

Dr. Percival Bailey presented two cases of metastatic tumor nodules in the left cerebral hemisphere causing aphasic syndromes. He gave first a review of Marie's conception of aphasia, especially the conclusions drawn from a study of war wounds. The cases were presented and it was shown how the use of Marie's schema enabled

in these two cases an accurate diagnosis to be made of the location of the tumor nodules. Dr. Bailey insisted upon the usefulness of Marie's conception to the neurologic surgeon. The paper will be published in full in the Archives of Neurology and Psychiatry.

Discussion: Dr. E. W. Taylor: We should be very much indebted to Dr. Bailey for his admirably lucid discussion of an always difficult subject. He has brought out in the fewest possible words the evolution that has taken place in our understanding of aphasia since the early days of Broca, through the monumental work of Marie, to the recent studies and investigations made by Henry Head. Those interested in teaching aphasia have been impressed for many years with the difficulty of the subject, and especially with the inaccuracy of the representation of sharply defined cortical areas. The ideas of anarthria and true aphasia as maintained by Marie, and that there is no such entity as motor aphasia such as Broca described, has been rather generally accepted. Cases of the sort reported by Dr. Bailey, which demonstrate the possibility of arriving at a perfectly definite diagnosis, and substantiating that diagnosis by a clean-cut operation are, of course, of the greatest possible significance. In the first case, I was rather interested in that the diagnosis was made rather definitely in the region of the supramarginal gyrus rather than in the zone further forward—I presume from the fact that there was no hemiplegia. One other point: Dr. Bailey said nothing about a so-called graphic region. I should like to ask whether or not he would accept as of diagnostic importance disturbance in writing, localized somewhere anterior to the base of the second convolution where he placed his second tumor.

Dr. Bailey, closing: Dr. Taylor wanted to know why in the first case I did not make a diagnosis in some other region than the supramarginal gyrus. Because the patient had a bilateral apraxia. If a patient has apraxia, one must assume involvement of the supramarginal region. Agraphia cannot be depended upon as of localizing value.

THE NEED OF A NEUROPSYCHIATRIC DEPARTMENT IN GENERAL HOSPITALS OF ONE HUNDRED OR MORE BEDS

DR. MABEL D. ORDWAY

In view of the fact that expansion and unification of knowledge are in these days recognized as the firmest foundations for progress, it is pertinent to bring before a society primarily devoted to scientific advancement the statement of an effort toward spreading the knowledge at hand, in the form of a statistical report presented at the October Clinical Conference of the New England Hospital for Women and Children.

"There is nothing physically wrong with this man, but he is terribly nervous and he is referred to your department for reassurance." This was the substance of a note from the physician-in-chief of one of the best and largest general medical clinics in Boston a few

years ago. He was referring the patient to the Nerve Department of the same hospital. The neurological diagnosis was "Advanced Multiple Sclerosis"—"Nothing physically wrong" and yet, extensive lesions in the brain and spinal cord.

We are indebted to the recent epidemics of encephalitis for the increasing interest of general medical men and women in neuropsychiatric conditions.

It has seemed of interest to collect a series of cases occurring in a general hospital (O.P.D. and house patients) where for two years the neuropsychiatric cases have been referred to a special department for the first time in the fifty years' existence of the hospital. No effort has been made to attract cases to the department and merely the cases which would have gone to the general medical department have, in part, been seen.

The following is a list of the first one hundred cases referred to the Out-Patient Department. The classification is the one used in the other hospitals in the city.

	Per cent
I. Brain.	16
II. Cerebrospinal (nonspecific)	1
III. Mental affections:	
1. Psychoneuroses:	
a. Neurasthenia	21
b. Psychoesthesia	9
c. Anxiety psychosis	12
2. Psychoses	7
3. Mental defect	8
IV. Miscellaneous:	
1. Chorea	4
V. Myopathies	2
VI. Peripheral nerves	5
VII. Neurosyphilis	15
	<hr/>
	100

In addition there were one hundred cases not referred but many of them were seen in consultation. The following is a list of these cases:

	Per cent
I. Brain	2
II. Cerebrospinal	0
III. Mental affections:	
1. Psychoneuroses:	
a. Neurasthenia	43
b. Hysteria	2
IV. Miscellaneous:	
1. Chorea	8
2. Masturbation	2
3. Neuroses	1

V. Myopathies :	
1. Myalgia	3
2. Myositis	1
VI. Peripheral nerves :	
1. Neuritis (single nerve)	6
2. Neuralgia	3
3. Multiple neuritis	1
4. Facial paralysis	2
5. Torticollis	1
VII. Glandular :	
1. Hyperthyroid	3
2. Parathyroid	2
3. Hypothyroid	1
4. Hypopituitary	2
5. Endocrinopathy	2
VIII. Neurosyphilis (?)	15
	<hr/> 100

One hundred and fifty of the house cases could be placed under a neuropsychiatric classification :

	Per cent
I. Brain	24
1. Cerebral hemorrhage	9 cases
2. Hydrocephalus	9 cases
3. Arteriosclerosis	7 cases
4. Embolism	1 case
5. Encephalitis	6 cases
6. Epilepsy	5 cases
II. Cerebrospinal	8
1. Infantile paralysis	1 case
2. Meningismus	1 case
3. Meningitis	6 cases
4. Spina bifida	4 cases
III. Mental affections	20
1. Psychoneuroses :	
a. Neurasthenia	11 cases
b. Psychasthenia	7 cases
c. Anxiety neuroses	1 case
d. Hysteria	1 case
2. Mental defect	7 cases
3. Psychopathies	1 case
4. Psychoses	2 cases
IV. Miscellaneous	20
1. Convulsions	6 cases
2. Aphasia	1 case
3. Cephalalgia	1 case
4. Chorea	16 cases
5. Neuroses	2 cases
6. Tetany	4 cases

V. Peripheral nerves	10
1. Torticollis.....	2 cases
2. Paralyzes	5 cases
3. Neuritis	7 cases
4. Neuralgia	1 case
VI. Myopathies.....	0
VII. Glandular	7
1. Endocrinopathy	1 case
2. Hypopituitarism.....	2 cases
3. Hyperpituitarism.....	1 case
4. Hypothyroidism.....	5 cases
5. Hyperthyroidism.....	1 case
VIII. Syphilis.....	11
1. Neurosyphilis.....	3 cases
2. Neurosyphilis(?).....	13 cases
	<hr/> 100

These cases were from the Maternity, Surgical (adult and children) and Medical (adult and children) Departments. This review of neuropsychiatric conditions appears to be ample justification for the existence of a department devoted to their consideration in hospitals of this size and character. Whereas the stimulation of the large hospitals is indispensable in arousing the interest of students and of young physicians, the personal atmosphere of the small hospital affords a remarkable opportunity for that type of mature mind which finds its chief interest in the synthetic study of the individual in the light of human relationships.

Discussion: Dr. C. M. Campbell: I hesitate to speak because it looks as if I were trying to reinforce what we are preaching all along, and I think it much more gratifying when the internists or those not most interested in psychiatric problems appeal to us for help, whereas if we go to them and offer to help, it looks as if we were foisting ourselves on them. We realize that of those who go to the general hospitals and to other specialists with medical or surgical disorders, an extraordinary per cent have disorders with an emotional, personal, or psychiatric origin. In the organization of the work of the community, it is very important to emphasize the point Dr. Ordway has emphasized: not to have special hospitals, but to see that in every hospital of fair size these problems of very grave importance are looked after in a special way, with decent consultation facilities, just as hospitals have fair facilities for other consultations. And much more important than having a special psychopathic hospital where patients can be transferred from general hospitals is the installation in every general hospital of a psychopathic department, and of course more important still is to inculcate in every internist a psychiatric standpoint, so that there will be in the mind of the consultant or practitioner, the physician and surgeon, the possibility of this avenue of approach to patients not complaining of mental or nervous trouble but of the ordinary ailments for which patients go to a physician.

EVIDENCE OF THE EFFECTS OF THE ENVIRONMENT
UPON GERM PLASM

Dr. A. Myerson: The prevailing doctrine in biological science which relates to the transference of qualities from one generation to another is the Weismannian doctrine. Weismann himself stated that the environment could injure germ plasm so that defective individuals might arise. The interpretation of his doctrines received in recent years, and especially in psychiatry and eugenics, has been that the germ plasm is almost inaccessible, that variation rises fortuitously, and that on the whole environment influences germ plasm but very little if at all. Much discussion in biological science has concerned itself with the question—"Can acquired characters be inherited?" But from the standpoint of medicine this is an academic question and what our profession needs to concern itself with is a more specific question, which is the following: "Can the environment influence the germ plasm so that a pathological process may become started in the germ plasm of one generation and continue, without renewed injury, from generation to generation?" *A priori* this would seem to be possible. Germ plasm is no metaphysical substance residing in the organism, in a sacro sanct chamber. It is the graffian follicle of the ovary in its evolution towards the ovum, it is the cell substance of the testicle on its way to become the spermatozoön, it is bathed by the same blood stream which bathes the rest of the organism, it has definite relations to the lymph stream and, in short, lives and dies with the organism.

This *a priori* opinion seems to me to be amply proven by recent experiments.

A. The work of Stockard, Craig and Papanicolaou. Guinea pigs were injured by the fumes of alcohol and defect of all kinds appeared in their descendants. This defect was transmitted for several generations, four at least, without any renewed injury by alcohol. A portion of the guinea pigs thus produced became progressively more defective so that this group disappeared, being unable to procreate. The other portion of these guinea pigs recovered its energies after a few generations and bred normally.

B. Experiments of Manfred Frankel. "X-ray treatment on the belly of very young guinea pigs produced not only a retarded growth in the treated animal, but in increased grade on the descendants of this animal through several generations, to the final result that by these later animals only one pregnancy comes to pass until finally the last generation remains entirely sterile." This type of research has been duplicated by others, notably, C. C. Little of this country with similar results.

C. The Work of Guyer and Smith. Perhaps the most spectacular and interesting experiments of all have been carried on by the Americans Guyer and Smith. Guyer and Smith injected into fowls the pulverized lens of rabbits. They then obtained a hen serum having a remarkable lytic or destructive effect upon the lens of rabbits. When this serum is injected in pregnant rabbits during the first 10 to 13 days of the pregnancy important results are obtained. Many

of the foetal rabbits die. In 9 cases out of 61 of those who survived, the lens was small and more or less opaque. In some cases there was a marked reduction in the size of the eyeball, and in others a complete nondevelopment of the eye. The control rabbits injected with serum of untreated fowls showed no such modification. In other words, the blood of the fowl had obtained the property of producing a serum which passed into the placental circulation, injuring the developing rabbit in a specific manner, mainly producing injuries to the lens substance and to the visual apparatus.

The most important and the really significant fact follows: When this pathological character appears in rabbits, it becomes hereditary without further use of the serum. It has been transmitted for eight generations, and what is more important, continually tends to become more serious in the successive generations and to become more common in the members of each generation. Defective males when crossed with normal females not belonging to any of the injured families show the following results: the first generation shows normal eyes, but the females of this generation again crossed with defective males give birth to a certain number of young with degenerated eyes.

D. The work of Tower. Tower treated by heat and cold the immature forms of a beetle and obtained color changes in the nature organism of a definite type. These changes did not persist in the next generation. Treating in the same way, the mature organism with mature reproductive organs he obtained in the next generation color changes of a definite type, *and these persisted generation after generation* without further use of heat or cold.

I am not here citing the researches of many other workers such as McCarrison, Adami, Carl Weller, etc. What I wish to emphasize is this: that the environment in one form or another may penetrate the organism and influence the germplasm so that a dynamic process is set going in it, a process which shows more and more results in each generation. This is what one sees in many families with mental disease. There is thus a striking similarity between familial mental diseases and the experimental results here cited. We may conclude that germplasm tends to resist change but while conservative, it is not reactionary, and can be played upon and altered by environmental forces. It is true that the environmental influences or forces must be penetrative, must reach the germplasm through body tissues; but this does not alter the fundamental fact that it is reachable and alterable.

Discussion: Dr. E. W. Taylor: The insistence upon exactness of thought as emphasized by Dr. Myerson is certainly of the utmost importance in dealing with the problem of heredity. Medical men have been flagrant offenders in this regard. The use of such terms as "good" or "bad" heredity in connection with a family history is meaningless and misleading. If any data of value are to be collected in the future, it is clearly necessary that such vague terms be avoided and an effort made to describe in detail the disorders from which the ascendants suffered before considering their possible relation to the

descendants. It is clear that many conditions which may have led to marked deterioration or mental disturbance in previous generations can have no effect upon the descendants, or at least an effect which is as yet little understood and appreciated. As has recently been pointed out, the main effort for the present should be to collect all facts possible, but to postpone their interpretation until the laws of heredity are developed to a point which can make practical their application. At the recent meeting in New York of the Association for Research in Nervous and Mental Diseases, the sharp cleavage between the geneticists' point of view and that of the medical profession was conspicuous, to the detriment of the medical men. It was shown clearly that the Mendelian laws cannot as yet be applied rationally to human heredity except in a very few instances, and that much of the ordinary discussion of heredity on the part of physicians is meaningless from a strictly biological viewpoint. In the meantime, more investigation and observation, and fewer premature deductions are desirable.

Dr. Myerson, closing: In every medical hospital the question arises, Has some ancestor had tuberculosis, cancer, or insanity. We have found that in the thousands of cases in which insanity has been reported, that statement is worth absolutely nothing. In the first place, alcoholic diseases occur more frequently in the ancestors of the so-called sane than of the insane. Hemiplegia is rather normal, and if a descendant of a person with hemiplegia has dementia precox, there is no relation between the two. All the Mendelian work that has been done on mental disease is worthless and worse than that. What I am pleading for is that in every hospital taking the family history of cases, the hereditary history should be stated in detail; that the type of mental disease or nervous disorder be analyzed; that no blanket term be allowed at all; and that experimental work be done on heredity; that it is the field for the psychiatrist and not for the biologist.

CURRENT LITERATURE

I. VEGETATIVE NEUROLOGY.

2. ENDOCRINOPATHIES

Guttmacher, M. S., and Guttmacher, A. F. MORPHOLOGICAL AND PHYSIOLOGICAL STUDIES ON THE MUSCULATURE OF THE MATURE GRAAFIAN FOLLICLE OF THE SOW. [Johns Hopkins Bulletin, December 1921.]

In the morphology of the ovary Corner, Robinson and others have pictured smooth muscle cells, as a constituent of the theca externa of the mature Graafian follicle. Through both histological and physiological studies the authors have been able to prove definitely the existence of this muscular tissue in the sow. The Von Gieson technique demonstrated these muscle fibers morphologically. Following the usual *in vitro* technique the contractility of this element in the wall of the theca externa was produced by barium chloride stimulation. An investigation of the nervous mechanism of the follicle was undertaken to determine if possible the functional relation of the follicular nerves to this muscular element. In a review of the extensive literature on the innervation of the follicle one was confronted with a diversity of views as to the depth of penetration of the nerve fibers and their relation to the follicle. No comprehensive study was found by the authors on the sympathetic and parasympathetic balance of either the follicle or the ovary. It is very difficult to stain satisfactorily the nerves of the Graafian follicle. The delicacy of the fibrils makes microtome sections of the material quite unsatisfactory, for at best the sectioned nerve fibers look like a series of fine dots of doubtful structure. It was apparent to us therefore early in our study that the information that we sought could best be obtained from gross material suitably stained. Good results were obtained by Ehrlich's *intra vitam* methylene blue stain following the modifications of J. G. Wilson. In the theca externa, the muscle containing layer of the follicle, there is an abundant plexus of nerve fibers. The nerves run, 10 to 15 together, in good sized nonmedullated bundles, independent of the perivascular network which was demonstrable in our preparations. These bundles of nonmedullated nerve fibers give off numerous fibrils to the surrounding tissue. The fibrils have the ordinary morphology of the nonmedullated nerve. These nerve fibrils terminate on muscle cells in sympathetic motor endings, the smaller endings covering the muscle cell, while the larger terminations overlie several. These endings were of two morphologically contrasting types—the one large, pale, clear and oval, while the other is small, dark, full of granules and triangular in

form. We have therefore a complete path: nerve fibrils running intimately among muscle fibers, sympathetic motor endings on muscle cells, and muscle cells capable of functioning. It seems unquestionable that there must be some physiological application for this apparatus. Pharmacodynamic studies also were carried out on the follicle wall to ascertain the type of autonomic innervation. In this work we early noted that the smooth muscle of the Graafian follicle wall is very sensitive to a variation in the hydrogen-ion concentration either to the alkaline or acid side. On the stimulation of the true sympathetic (thoracolumbar) nerves by the addition of chemically pure epinephrine, furnished us by J. J. Abel, a definite relaxation of the follicle was recorded. However, on addition of a parasympathetic (bulbosacral) stimulant such as physostigmine sulphate (eserine) the follicle wall actively contracted. The proof of the parasympathetic innervation was further substantiated by the action of atrophine sulphate, which paralyzed the myoneural junction of the parasympathetic nerve fibers, so that on the addition of physostigmine no contraction was elicited; atrophine also relaxed the tissue previously contracted by physostigmine. It is then obvious that the follicle has an antagonistic autonomic innervation, similar to the intestines, the true sympathetics acting as inhibitory and the parasympathetic as excitatory nerves. A similar innervation was found for the ovarian stroma. [Author's abstract.]

Maranón, G. HYPOGENITAL HAND. [Siglo Médico, July 1921, LXVIII, No. 3527.]

This author describes a syndrome affecting the development of the human hand which he relates to inferiority of the gonadal system. He terms it the *hypogenital hand*. It is characterized by its fat, puffy, cold, clammy, acrocyanotic appearance and feel. The vasomotor disturbances inducing the acrocyanosis are traceable to insufficiency of the genital glands, predominantly of the ovaries. There may be insufficiency of other endocrine glands, but the genital insufficiency is predominant and constant. A glimpse of the congested cyanotic, clammy hands is enough to suggest genital infantilism; the hands are usually puffy and doughy, and the nails are often spotted. The age is between puberty and maturity or at the menopause, and the subjects are usually females. Time and organotherapy are the reliance in treatment, especially intensive and prolonged ovarian treatment.

II. SENSORI-MOTOR NEUROLOGY.

4. MIDBRAIN—INTERBRAIN.

Morquio. EPIDEMIC ENCEPHALITIS. [Arch. Lat. Am. d. Ped., November–December 1920, XIV, No. 6.]

This author describes some cases with clear cut choreic syndromes, and further suggests that Sydenham's chorea may be regarded in part as related to the epidemic encephalitis picture. Is it a disease of sporadic

occurrence, always the same, or are there different entities influenced by special etiologic conditions? We must not forget, he adds, that infantile paralysis has had to yield to the pressure of facts which have demonstrated the existence of a specific virus capable of originating epidemic forms which we know to-day as epidemic poliomyelitis.

Netter. HERPES IN LETHARGIC ENCEPHALITIS. [*Bull. et Mém. Soc. Méd. des Hôp. de Paris.*]

This author calls attention to the experimental researches of Doerr, Vochtlings, and Schnabel, Blanc and Caminopietro, Levaditi, Harvier, and Nicolau, Lueger and Lauda, Netter, Cesari, Mawas, and Salanier which tend to show the existence in the fluid of herpetic vesicles of a virus capable of producing symptoms and lesions identical with those produced by inoculation of the rabbit with the virus of encephalitis. Doerr and Schnabel, and Levaditi and his collaborators have shown that rabbits which have resisted inoculation with the virus of encephalitis are refractory to the virus of herpes and vice versa. Levaditi has even succeeded in producing cutaneous lesions by local application of the virus of encephalitis. Contrary to what might be expected, however, herpes is rare in lethargic encephalitis. Out of 180 patients observed by Netter during the acute stage herpes occurred in only two—in one on the face and in the other on the thigh. In 94 cases reported in detail in the *Bull. et Mém. Soc. Méd. des Hôp. de Paris* in 1920, herpes was mentioned only once, and in only one of the 223 reported by McNalty was herpes noted. American and Italian writers also state that herpes is rare in encephalitis. [B. M. J.]

Cawadias, A. EPIDEMIC ENCEPHALITIS IN GREECE. [*Compt. Rend. Soc. de Biol.*, January 22, 1921, LXXXIV, pp. 137, 139.]

An epidemic of encephalitis began in Greece in January, 1920. Its origin is uncertain, but a few months previously some cases of febrile chorea, difficult to classify, were seen. A notable feature of the epidemic was the predominance of excitation-forms such as the myoclonic and delirious, etc. There was no purely lethargic epidemic, but at the outset a polymorphic epidemic with predominance of myoclonic forms. A case is cited of a marked meningeal reaction without lymphocytosis of the cerebrospinal fluid—somnia, slight fever, ptosis, internal strabismus, and a positive white line. A week later the temperature rose a little, with bilious vomiting and opisthotonos; a week later, convalescence. Another case showed myoclonic movements with generalized pruritus. A child of twelve years had general malaise, high fever, and vertigo. Three days later, delirium and intense generalized myoclonic shocks; a few days later there was severe generalized pruritus with rhythmical muscular shocks of the diaphragm—observed by X-rays—and at the same time of the abdominal muscles. There also was violent dreamy delirium and retention of urine. The result of the case is

unknown. In general, Cawadias found that polymorphism was characteristic of this Greek epidemic of encephalitis.

The blood in these cases showed slight hyperleucocytosis, with 70-80 per 100 of polynuclears. Urea in the blood 0 gr. .50 to 0 gr. .70. Hemoculture negative. Never any marked reaction in the cerebrospinal fluid, 2 to 3 lymphocytes in a field, once ten; in one case none. Albumin always increased. Glucose not increased. Chlorides definitely increased; urea also greatly increased in the spinal fluid. Thus there has been in these cases absence of lymphocytosis with definite increase, at the height of the disease, of the chlorides, urea, and albumin in the cerebrospinal fluid, but never any hyperglycorrhachie. [Leonard J. Kidd, London, England.]

Girardi. SEQUELÆ OF ENCEPHALITIS LETHARGICA. [Il Morgagni, February 28, 1922.]

The author speaks of the grave prognosis to be borne in mind in all cases. Many cases, dismissed from hospital as cured, relapse after a brief period of good health—for example, out of 13 cases discharged as cured in 1919-20, 8 returned in 1921 with a recurrence of symptoms. Very commonly the symptoms are like paralysis agitans (dull monotonous voice, rigidity, expressionless face, tremor, etc.), and the more striking, as the patients are usually much younger than the age at which one sees true paralysis agitans. The muscles are easily tired and general asthenia is one of the commonest symptoms—inability to stick at their work, soon tired. Arranging the relative frequency of the sequelæ in a graphic form, it is shown that in order of frequency nystagmus heads the list, followed by pseudoparalysis agitans, myoclonus disturbances of sleep (in children often taking the form of semidelirium), ptosis, facial asymmetry, recurrence of initial symptoms, diplopia, choreiform movements, and lastly, disturbances of respiration. The cerebrospinal fluid showed no pathological changes. As contrasted with the tremor of true paralysis agitans, the tremor in pseudoparalysis agitans is increased in movements and is not rhythmic. True paralysis agitans has been known to follow encephalitis.

Abadie and Hesnard. POST-ENCEPHALITIC PARKINSONIAN ATTITUDE. [Gaz. Hebd. des Sci. Méd. de Bordeaux, 1921, XLII, 43.]

The writers report to the Bordeaux Society of Medicine and Surgery the case of a boy of fourteen who in January-February, 1920, had severe acute epidemic encephalitis with some discrete meningeal symptoms and with lymphocytosis and 0 gr. .80 of albumin in the cerebrospinal fluid. Now, nine months after convalescence from his acute illness, he has a stiff attitude and general aspect of fixity, with mask-like face, everted lips, frequent prolonged spastic smiling, and dribbling of saliva. He sits bent to one side and stiff, with hands in a Parkinsonian attitude; he carries out commands, but extremely slowly; at times he has tremor

in his upper limbs; gait slow, all in a piece; latero- and retro-pulsion marked; slight stiffness of muscles on passive movements, but no true contracture; speech monotonous, distant, nasal, sometimes tremulous; slight electrical hypoexcitability of both facial nerves; bilateral incomplete palsy of accommodation, crossed diplopia, paralysis of convergence; no psychical symptoms; general state middling. The writers emphasize the interest of this attenuated but persistent Parkinsonian attitude after nine months. The post-encephalitic Parkinsonism may (exceptionally) pass into true paralysis agitans, or become fixed, or slowly disappear. They think that the clinical picture in this case may be explained as being a sequel of an encephalitis localized in the globus pallidus of the striatum and the neighboring nuclei, and in the locus niger. [Leonard J. Kidd, London, England.]

Amoss, H. L. IMMUNOLOGIC DISTINCTION OF ENCEPHALITIS AND POLIOMYELITIS. [Jour. Ex. Med., February 1, 1921. J. A. M. A.]

Experiments made by Amoss show that the two diseases can be distinguished through the power of blood serum under certain circumstances to neutralize the virus of poliomyelitis. The blood serum of convalescent cases of poliomyelitis whether in man or monkey possesses this neutralizing power, while the blood serum of recently convalescent cases of epidemic encephalitis is devoid of it. On the basis of the distinguishing characters described, it is regarded as desirable at the present time to hold epidemic poliomyelitis and epidemic encephalitis as integrally distinct affections. The latter also may be infectious, yet the main lesions of poliomyelitis are present in the spinal cord, and of epidemic encephalitis in the midbrain.

Sanz. PROGNOSIS OF EPIDEMIC ENCEPHALITIS. [Arch. de med., cir., y espec., February 11, 1922.]

The mortality of epidemic encephalitis varies considerably with the time and place in which the disease has been studied. According to Banús it ranges from 25 to 31 per cent; in Spain, however, it is approximately only 10 per cent; according to Tilner and Howe it was 25 per cent among their 20 cases. Chalié, on the other hand, estimated it as high as 40 or even 50 per cent, and attached an unfavorable significance to the progressive evolution of myoclonic movements. The mortality in Sanz's cases was 17.64 per cent. All these figures refer to the acute stage of the disease. In the chronic stage the danger to life is less, but the prognosis is unfavorable, owing to the persistence of symptoms which interfere with the motor activity of the patient. Moreover, there is always the possibility of the recurrence of the principal symptoms, though this is a fairly uncommon event. The motor sequelæ tend to subside slowly with alternate recrudescences and remissions, but in some cases persist indefinitely and defy all methods of treatment. In addition to the Parkinsonian syndrome, which is the most frequent sequel

and one of the most refractory to treatment, Sanz mentions a cerebellar form of disseminated sclerosis and a chronic serous meningitis as sequelæ of epidemic encephalitis. Finally, as an aggravating factor in the remote prognosis of epidemic encephalitis, there should be mentioned the state of depression into which the patients fall owing to the apparent hopelessness of their condition, with the result that some of them show a tendency to suicide. [B. M. J.]

Anglade. PATHOLOGY OF EPIDEMIC ENCEPHALITIS. [Gaz. hebdomadaire de Médecine, January 16, 1921.]

The pathological picture here recorded is as follows: (1) the frontal cortex was always the site of a process of encephalitis. The large and moderate sized nerve cells presented various degrees of chromatolysis. (2) The grey substance of the cord showed changes similar to those observed in the cortex. (3) The lesions were diffuse in both. Epidemic encephalitis is, according to the author, the result of a general infection. Histologically this infection is manifested (1) by disseminated inflammatory lesions, (2) by focal lesions situated around vascular terminations. The mesencephalon, grey substance at the base or center of the brain, and the locus niger appear to be the sites of predilection for the formation of these foci.

MacLaire, A. S. DELAYED DIAGNOSIS IN A CASE OF EPIDEMIC ENCEPHALITIS. [Neurological Bulletin, March 1921, Vol. III, No. 3.]

The author reports the case of a man, twenty-eight years of age, who at the time of examination complained of nervousness, weakness, insomnia, stiffness and tremor for a period of four months. Seven months before the first interview, the patient began to suffer with severe headaches and burning sensations in the head, varying in position from the top to the sides and back of the head and neck. The intensity of the cephalalgia was such as to prevent sleep. A tremor gradually developed in the right hand and leg which was present both at rest as well as in motion. General rigidity and weakness was next noticed. As the tremor and rigidity increased the headaches and insomnia ceased. He then became easily fatigued and his speech became monotonous and slow. The physical examination revealed mask-like features and general rigidity of the body. There was a loss of the associated movements of the right upper extremity. Speech was slow and tone lowered. A coarse, medium tremor of the hands was present, the right greater than the left, intensified by motion and absent when at rest. Tremor in the lower extremities was also present, the right being greater than the left. There was a coarse, rigid tremor of the lips when the tongue was protruded, also a fine tremor of the tongue. Slight cogwheel release on the right side at times, more marked in the leg. Muscular movements were rigid. It was a difficulty for him to wrinkle his forehead. Laboratory findings were all negative including tests for

plumbism. Here was a man who had gone seven months before a correct diagnosis had been made. Previously paralysis agitans, *per se*, was not commonly met. The possibilities considered in the diagnosis were true Parkinson's disease, metallic encephalitis and paralysis agitans type of epidemic encephalitis. A diagnosis of the last possibility was made on the basis of an acute infectious illness during an epidemic period with a residual paralysis agitans syndrome which was a common finding. To state that a plea is made to the profession for carefully detailed histories is a bromide to say the least. Yet if one reads the histories taken, those that are, by many physicians one is not at all astonished to see cases drift along from one doctor to another without a proper diagnosis being made. History taking is a science in itself and when conquered is an asset for the possessor. At least fifty per cent of the diagnosis depends upon the history. It is only after a diagnosis is made that rational therapy can be instituted. Symptomatic treatment and placebo medication when not justly indicated is charlatan practice and should be relegated to the medieval days. Thus in closing, the author wishes to be placed on record with the large foregone group who have advocated careful history taking. For, if we were all equally equipped, as we should be, there would not be any necessity for reporting "A Case of Delayed Diagnosis in Epidemic Encephalitis." [Author's Abstract.]

Alfaro. EPIDEMIC ENCEPHALITIS AND CHOREA. [Arch. Lat.-Am. d. Ped., November-December 1921, XV, No. 6. J. A. M. A.]

Aráoz Alfaro argues that the facts observed in epidemic encephalitis have demonstrated that ordinary chorea in the majority of cases is a mild encephalitis, localized mainly in the striate body, and due to various infectious agents acting on an unstable nervous system, constitutionally or temporarily substandard. The infectious agent most commonly involved is the one that is responsible for acute articular rheumatism. Other infectious and toxic agents are also liable to induce it in the predisposed, and intense emotional stress may be the occasional cause.

Barker, L. F., and Sprunt, T. P. TETANY AND HYPERPNEA IN A PSYCHONEUROTIC PATIENT CONVALESCENT FROM EPIDEMIC ENCEPHALITIS. [Endocrinology, VI, 1922, 1.]

The patient, a young man of eighteen years of age, gave a history of an acute attack of encephalitis one year before. This was characterized by sudden onset, fever, diplopia and maniacal delirium for two weeks. He had always been nervous and was the only remaining child of solicitous parents. The residuals of the encephalitis included increased nervousness, periods of very deep breathing, lethargy during the day and nervousness and insomnia at night, trembling spells, and occasional lapses into a slightly manic state with partial amnesia. Neurological examination revealed no signs of organic disease. During the first

examination, the patient was excited and the deep respirations were unusually marked and prolonged. To this fact was attributed the typical attack of tetany with carpal spasm, positive Chvostek sign, twitching of facial muscles, paresthesias of hands and arms and profuse sweating of the face and neck. The duration of the attack was about fifteen minutes. Preceding the examination during which this attack of tetany occurred, the patient had shown no signs of latent tetany and two days later these signs were negative and the electrical excitability of the motor nerves was normal. Experimental physiological studies by Collip and Backus (*Am. J. Physiol.* [Balt.], 1920, LI, 568) and by Grant and Goldman (*Am. J. Physiol.*, 1920, LII, 209) had shown that tetany regularly occurs in normal individuals as a feature of voluntary prolonged hyperpnea, probably as a result of disturbed acid-base equilibrium in the body. Having the tetany particularly in mind, Grant and Goldman conducted a series of about twenty experiments and were able to produce tetany in each instance after the deep breathing at the rate of about twelve per minute had been continued for from thirteen to seventeen minutes. Their experiments were carefully controlled on the chemical side and the tetany was found to occur after decided changes had taken place in the alveolar air, in the blood and in the urine. The alveolar carbon dioxide tension fell, the blood became slightly more alkaline, the urine became decidedly alkaline, the plasma bicarbonate was reduced, the ammonia excretion was diminished, and there was a slight increase in the calcium content of the blood.

Vincent, M., and Gaujoux, E. EPIDEMIC ENCEPHALITIS IN THE PREGNANT. [*Revue Franç. de Gynécologie et d'Obstêt.*, March 1921, XVI, No. 3. J. A. M. A.]

Vincent and Gaujoux describe a case and summarize eleven others from the literature, and urge the importance of laboratory tests to eliminate other infectious processes. They verify the diagnosis by the mild meningeal reaction or high sugar content of the spinal fluid which is so frequent in this disease. The pregnancy in itself does not seem to render the prognosis graver.

Senise, T. LAUGHING AND SPASTIC LAUGHING IN POST-ENCEPHALITIC PARKINSONIAN SYNDROMES. [*Il Cervello*, January-February 1922, I, 1,]

The author in studying the mimic disturbances found in some cases of Parkinsonian syndrome consecutive to epidemic encephalitis has made a special analysis of laughter. He calls the type of laughter found in these patients *rigid* to differentiate it from the *spastic* form encountered in some cases of lesion of the pyramidal system (myodynamic or corticomuscular system) such as in hemiplegia in congenital and acquired paraplegia, in lateral sclerosis, in pseudobulbar paralysis, etc. In contrasting the two forms of laughing, spastic and rigid, he says that the

latter is slow, forced, fixed, gentle, persistent, often incomplete, never loud and explosive. It is merely an aspect of the general rigidity found in Parkinsonian patients who, as it is well known, execute slow, gentle, forced movements (*bradykynes* of Marie and Lévy, cog-wheel phenomenon of Negro and Mayer). Once, however, these movements are produced they tend to persist in spite of the patient's will. This rigid laughing being the expression of an involvement of the extrapyramidal motor system (striate body, rubrospinal tract, etc: *myostatic system*), may be found also in Wilson's disease, in some forms of pseudo-bulbar paralysis of Parkinsonian type (Brissaud, C. and O. Vogt), etc.

The author, who is a gifted student of the psychology of laughter, has indicated the semiologic and physiopathologic differences of the two forms of rigid and spastic laughing, because some authors have mentioned the latter forms in Parkinsonian patients. The crying in these individuals has also the same special characteristics (rigid crying) which differentiates it from the spastic crying. [Naccarati, New York.]

Sainton, P., and Cornet, P. SIGNS OF EPIDEMIC ENCEPHALITIS. [Paris Médical, May 1921, XI, No. 21.]

Sainton and Cornet have found it possible to elicit myoclonus in dubious cases by flexing the hand or arm or tapping certain muscles. Another sign is what they call the frontal sign. When the upper eyelid is raised, the frontal muscle contracts very slowly. In exophthalmic goiter it does not contract at all, and in seven patients with epidemic encephalitis this asynergy in the movements of the upper lid and the frontal muscle was pronounced.

Hassin, G. B. HISTOPATHOLOGIC FINDINGS IN A CASE OF SUPERIOR AND INFERIOR POLIENEPHALITIS WITH REMARKS ON THE CEREBROSPINAL FLUID. [Am. Archives of Neurology and Psychiatry, May 1921, Vol. V, pp. 552-567.]

A twenty year old man complained of headache, vomiting, difficulties in swallowing and speech troubles (dysarthria). The examination revealed paralysis of the third, fourth, fifth, sixth, seventh, ninth and twelfth cranial nerves, and a partial involvement of the tenth and eleventh. The reflexes, sensibility, genito-urinary organs, spinal fluid were normal. Death was sudden two months after the onset of the bulbar symptoms.

The microscopic examination of the brain showed a widespread degeneration of the gray matter, especially of the midbrain and medulla. The degeneration was associated with marked vascular hyperemia, mild proliferation of the capillaries and glia changes. The latter showed as cytoplasmic glia, myelophages packed with remnants of myelin and axones, and various forms of gutter, or fat granule, cells loaded with lipoids. The nerve fibers were swollen or tumefied, and broken up into small fragments. The pia archnoid appeared distended, the meshes filled

with lymphocytes, mesothelial cells and a great number of fat granule bodies which were also found in the choroid plexus. This was packed with fat-like substance contained within the enlarged vacuolated tuft cells.

The microscopic findings were typical for secondary nerve degeneration and the condition can not, therefore, be designated as encephalitis. It is a degenerative process analogous to that seen in amystrophic lateral sclerosis, multiple sclerosis, subacute combined cord degeneration and similar states. The presence of fat-like or lipid substances within the perivascular spaces and the subarachnoid space indicates that these formations drain the lipoids from the areas of degeneration, and that the contents of the subarachnoid space which in this case consisted mainly of lipoids come from the brain tissues. In short, the cerebrospinal fluid as derived principally, if not exclusively, from the tissue fluids of the brain.

Another noteworthy feature, the presence of lipoids, in large quantities, within the tuft cells of the choroid plexus, indicates that the latter derives the fat from the cerebrospinal fluid, that the spinal fluid before being absorbed by the usual channels is purified by the choroid plexus. The function of the latter is probably to pick up from the cerebrospinal fluid harmful or other products and to render them as well as the fluid itself, more absorbable. [Author's abstract.]

Gamma. CORPORA AMYLACEA IN EPIDEMIC ENCEPHALITIS. [Arch. p. 1. Sc. Med., 1921, Fase. 1-2.]

Numerous Italian observers have reported the occurrence of corpora amylacea with strikingly increased frequency in the central nervous system of patients who have died of encephalitis epidemic. Some have even attributed to this increase a significance comparable to that of the Negri bodies in hydrophobia. Gamma finds that even in young subjects the corpora amylacea in cases of epidemic encephalitis are extremely abundant, considerably exceeding in frequency those found in disseminated sclerosis and other chronically progressive maladies of the central nervous system. In general their number is less in the cases taking a more acute clinical course. The chemical and tinctorial properties, although somewhat variable, do not differ from those of the corpora amylacea in general. Gamma is unable to confirm the finding of certain observers that the corpora amylacea of epidemic encephalitis show special staining properties with iodine and sulphuric acid. The corpora amylacea he finds to be particularly abundant in the white matter of the posterior regions of the cord, medulla, pons, midbrain, and centrum ovale; from their frequent occurrence in proximity to the cavities which contain cerebrospinal fluid, and from a study of their histological characters, he is inclined to regard them as a product of slow regressive changes in the nervous rather than in the neuroglial elements, and as taking a centrifugal course tending to their eventual elimination. The

more externally situated bodies, which are frequently found near the blood vessels, are in general larger, more dense, more deeply staining, and presumably of less recent formation. [B. M. J.]

Guillain, G., and Gardin, C. PARKINSONIAN SYNDROME WITH EPIDEMIC ENCEPHALITIS. [Bul. de la Soc. Méd. des Hôp., May 1921, XLV, No. 16.]

These authors record a case with high blood pressure, glycosuria and catatonic muscular rigidity in an epidemic encephalitis case.

Piotrowski, G. EPIDEMIC ENCEPHALITIS. [Schweiz. Arch. f. Neurol. u. Psychiatrie, 1921, VIII, No. 2.]

This is an extensive résumé of some nearly 500 studies on encephalitis.

D'Antona, S. SYMPTOMATOLOGY OF EPIDEMIC ENCEPHALITIS. [Annali di Neurologia, 1921, XXXVIII, Nos. 1-2.]

This observer calls attention to the vegetative nervous system disturbances, particularly the vasomotor variations. The possibilities of vegetative neurological findings are insisted upon.

Livet, L. OBESITY AFTER EPIDEMIC ENCEPHALITIS. [Bull. de la Soc. Médicale des Hôp., May 1921, XLV, No. 15.]

A clinical report of three cases in which obesity developed following epidemic encephalitis. Organotherapy was of value and gave proof of the endocrine nature of the disturbance. In another case the relapses of the disease, after a year's course, were effectually combated by light baths, tepid douches and hexamethylenamin in 2 gm. doses by the mouth.

Kummer and Fol. ILEUS WITH MYOCLONUS. [Bull. de l'Académie de Médecine, May 1921, LXXXV, No. 19.]

The clinical abdominal signs in encephalitis are frequent. This paper calls attention to rhythmic jerking and twitching of the abdominal wall and diaphragm accompanying the occlusion of the bowel in the woman of forty-nine. At first they seemed to be a form of epidemic encephalitis. Acute malignant nephritis was discovered as the only cause.

Rosenheck, C., and Cornwall, L. H. ATYPICAL EPIDEMIC ENCEPHALITIS. [Neurological Bulletin, July 1921, III, No. 7, 259-264.]

Rosenheck and Cornwall have reported a group of six cases which they believe to be atypical forms of epidemic encephalitis. With the exception of the first case they were all ambulant, which is quite a variant from the usual symptomatology of epidemic encephalitis. Two of the cases presented abnormal pupillary phenomena suggestive, in a clinical sense, of neurosyphilis. One of these cases was subjected to an intensive course of salvarsan therapy as a result of which the symptoms were all exaggerated. The negative Wassermann reactions in the blood and spinal fluid of all cases and the negative histories together with the

mode of onset and variety of symptoms led to the exclusion of syphilis. One case, which is related in considerable detail, illustrates the intervals that elapse between the development of new symptoms. They suggest that this case illustrates a recrudescence of pathological lesions in new anatomic situations therefore resulting in new clinical pictures. They emphasize the clinical symptoms which were insomnia, emotional instability, very slight or no rise in temperature, the predominance of purely spinal phenomena such as intermittent dull boring pain of a vague shifting character, muscular sensitiveness, scalp pain, bladder disturbances, plegias, and alterations in reflex behavior. The necessity of examination of the spinal fluid for confirmatory diagnosis is emphasized. The pressure was increased and the cell counts were pathologic in all of their cases. Cornwall gives the opinion that the polymorphonuclear percentage in encephalitis is higher than in anterior poliomyelitis. The globulin was increased in four of the six cases. The quantitative determination of the sugar content of the spinal fluid is advised in cases suspected of being encephalitis. They point out that the colloidal gold curves in encephalitis are usually low and emphasize that this reaction has its chief value when correlated with other laboratory and clinical findings as similar results are obtained in neurosyphilis, anterior poliomyelitis, low-grade meningitis, epilepsy, alcoholic neuritis, multiple and lateral sclerosis, brain tumor, hemiplegia, chronic nephritis and eclampsia. They mention that the Vernes reaction was used with the Wassermann as a diagnostic adjunct. Although in the title these were described as atypical forms of encephalitis, the authors mention that encephalomyelitis or meningomyelitis are terms which more properly describe the cases. At this time it is fortunate to have attention called to cases of this kind in view of the unusual symptomatology which could be easily confused with neurosyphilis, ganglionitis, myositis or psychoneurosis. More frequent lumbar punctures in cases of this sort would probably identify many cases which present but few neurologic symptoms as low-grade infections of the neurospinal axis. Whether the etiologic agent is the same which produces acute epidemic (so-called lethargic) encephalitis remains to be determined. [Author's abstract.]

Lhermitte. PEDIGREE OF EPIDEMIC ENCEPHALITIS. [Gaz. des Hôp., January 8, 1921.]

As our knowledge of this affection slowly accumulates, including the inquest into the remote past, our conception of the pedigree becomes variously modified. At present the case of Albrecht von Hildesheim, 1695, since it was sporadic, should hardly be counted and the epidemic which was first recorded was that of Tübingen in 1712. The author then waives all alleged epidemic incidence until the time of Gayet in 1875 when this author described the pathogeny of the syndrome and showed that it must originate in inflammatory lesions grouped about the third ventricle and the aqueduct of Sylvius. Lhermitte now insists that

most of the cases described by Wernicke in 1881 under the name of polioencephalitis superior hemorrhagica were examples of lethargic encephalitis. In 1887 Gerlier's disease was described and in the author's opinion must have been a variation of the same malady. Next in chronological order comes the malady, "nona," 1889-90, which showed such an extraordinary polymorphism that the profession was divided in opinion as to its nature, some even holding that it was a massed psychogenic affection. The author cites seven medical men who described nona and showed clearly that it was not a legendary affection, but it remained obscure until studied in the light of our recent knowledge, when it is recognizable as epidemic encephalitis.

Passing by certain Italian cases in the interim, probably because of their sporadic incidence, the author comes to the outbreak of 1916. But while this appeared in the same general area as older episodes the outbreak of the malady in regions outside of Central Europe and especially in remote Australia gave it a new status. There it was a frankly mysterious affection and one *sui generis* from the start. But its first appearance in England passed for botulism. Reports of the disease in such scattered parts of the world lent some plausibility to the spoiled food theory—an accident coming in the wake of the war. One cannot but admit that true botulism appeared in the same manner in widely scattered foci. Already there were marked local distinctions in the pathology. Thus the Austrian cases appeared to show meningeal involvement, which was notably absent in the French cases. The broad distinction between the lethargic and myoclonic forms became apparent and in Germany the latter may have predominated from the start with unusual clinical expressions—for example in Munich there was a pseudotabes. Italy, which had been visited by "nona," showed notable immunity in the present outbreaks, and when the epidemic appeared in 1919 the early cases were lethargic followed by the opposed type. When the disease reappeared in Austria it had assumed the myoclonic type. This sequence was certainly the rule but the author quite ignores the claim of Cruchet of Bordeaux that he recognized the myoclonic form at a period long antedating the appearance in France of the lethargic cases. In summing up, the author states that between the epidemic episodes sporadic cases have always been in evidence and cites a long list of authors who have described these cases, including such authorities as Thomsen and Oppenheimer. [Ed. Med. Rec.]

Netter, Césari and Durand, H. THE SALIVARY GLANDS IN EPIDEMIC ENCEPHALITIS. [Bulletins de la Société Médicale des Hôpitaux, May 1921, XLV, No. 16.]

Sialorrhea has been so frequent in this syndrome that a special study of the salivary activity has suggested this paper. Several cases with necropsy findings in some are here reported. The virulence of the salivary glands by injection of guinea-pigs and rabbits is noted.

Holmström, R. EPIDEMIC ENCEPHALITIS. [Hygiea, April 16, 1921, LXXXIII, No. 7.]

A clinical analysis of the mental and sensory motor signs in 27 cases. Six died. One patient had a relapse of hallucinations and paresthesias six months after his initial attack. Another a paretic relapse of the arms four months after the onset. In treatment rest in bed and mental repose should be maintained as long as possible. Some of his patients had been sent to the hospital with the diagnosis of paratyphoid, ileus, influenza, polyneuritis, appendicitis, perforated gastric ulcer. All had fever, and there had been a longer or shorter period of somnolency in all, and also nystagmus, and the majority had diplopia.

Denyer, S. E., and Morley, D. E. MYOCLONIC ENCEPHALITIS LETHARGICA SIMULATING HYDROPHOBIA. [Br. Med. J., 1921.]

In the discussion on epidemic encephalitis in 1918, at the Royal Society of Medicine, Dr. Crookshank pointed out that the old physicians used to describe "hydrophobia without the bite of a dog," and that this condition had also been reported in England in 1918. The following case was one in which hydrophobia was thought to be a possible diagnosis.

A. B., female, aged twenty-five years, up to the morning of November 11, 1920, was in good health, and with the exception of three attacks of influenza had had good health for many years. On the evening of November 11th she did not feel well; on November 12th she gave up her usual occupation owing to increased feeling of illness. She was first seen by one of us (D. E. Morley) on November 13th, and appeared to be suffering from a mild influenzal attack with no special symptoms. When seen on the 14th she was distinctly excited and anxious about herself; she had vomited in the morning, and headache had become a more marked feature. On November 15th, when seen (by D. E. M.) she became, as she had been earlier in the day, maniacal. Sitting up in bed, she shouted that her mouth was paralyzed and that she was going to die. The left side of the face was weak but there was no definite paralysis. On the 16th she was wildly delirious, the most marked feature being dread of trying to swallow, and the prolonged laryngeal spasm caused by the attempt to do so. The same ideas of paralysis and death still pervaded her delirium. The condition was such as one would expect to find in a case of hydrophobia, only there was no reasonable cause for such a diagnosis.

On November 17th she had subsided into a semi-comatose condition and was seen in consultation (with S. E. Denyer). Encephalitis lethargica was diagnosed. The condition found (by S. E. D.) was as follows: Temperature 100°, pulse 88, good; limbs flaccid, no trismus, slight twitching of the facial muscles on the right side, pointing to irritation of the seventh cranial nerve. She also had occasional clonic movements of her arms. The knee-jerks were present, no Babinski, no

ankle clonus, tendo Achillis jerks not obtainable, no Kernig's sign. On stroking the foot fairly hard, irregular twitching of the muscles of the outer side of the thigh took place. There had been no incontinence of urine; the bladder was empty. Pupils react to light but are unequal, the left being slightly dilated. No nystagmus. She was in a lethargic state and did not answer questions or appear to be aware of anything that was going on. There was great difficulty in swallowing, owing to the marked laryngeal spasm. A fluid of purulent appearance was frequently regurgitated from the throat. There was subcutaneous emphysema at the root of the neck on the right side. *Tache cérébrale* was present. The abdominal superficial reflex was present. There was no squint or ocular paralysis, no cyanosis, no sweating, no rash, no retraction of the head or pain in moving the neck in any direction. She had been fed successfully with a nasal tube previously, but an attempt to repeat this set up a condition of spasm which made feeding impossible by this means. She was therefore given saline enemata containing sugar, and to these was added 40 grains of hexamine every eight hours.

This treatment was continued, and on November 19th 10 grains of hexamine was administered intravenously. On November 20th she was conscious, and a further 20 grains of hexamine was given intravenously. On November 21st she was much improved mentally, being conscious of her surroundings and able to speak sensibly, though her words were incompletely formed. Feeding now became possible by a stomach tube, though there was still considerable spasm until the tube was in position. Her general condition, however, became worse, and although the emphysema at the root of the neck which had been so marked on November 17th had completely gone, bronchopneumonia supervened and the illness terminated fatally on November 24th. The temperature varied from 100° to 102°, with the exception of rises to 104° and 105° on the third and fourth days of the illness.

Pipping. LETHARGIC ENCEPHALITIS IN CHILDREN. [Finska Läkarsällskapets Handlingar, November-December 1920.]

A clinical paper reporting a small epidemic of lethargic encephalitis among children six to fourteen years old. Between November 25th and December 9th he treated three cases in hospital and two others in a polyclinic. The most prominent symptoms were mental disturbances, headache, diplopia, fever, twitching of certain muscle groups, and insomnia which was succeeded by profound drowsiness lasting several days. The two polyclinic cases were comparatively mild, but in these, too, there was headache with fever, diplopia, restlessness, and insomnia succeeded by drowsiness. The muscular twitchings in one of the cases admitted to hospital were suggestive of severe chorea; the girl was maniacal and suffered from hallucinations. The source of infection could not be traced in any of these cases, which occurred sporadically, and in none was there

a history of symptoms indicative of influenza. It would seem that only a small minority of the community is susceptible to this disease, for it does not break out wholesale in institutions or affect more than one member of a family. Thus, in no case did other members of the families of these patients develop the disease, and in the children's home, from which one of the patients came, there were no similar cases. [B. M. J.]

Mongini, Silvio. AMAUROSIS IN EPIDEMIC ENCEPHALITIS. [La Riforma Medica, 1921.]

A case of a woman who, on November 2, 1920, was attacked by a feeling of illness with slight rise of temperature which lasted two days accompanied by headache. After two days she suddenly complained of diminution of vision in the right eye. The right amblyopia increased rapidly so that on the third day from the beginning it gave place to complete loss of the visual function of this eye. The vision of the left eye remained intact. But three days later amaurosis of the right eye was established and in a complete and unexpected manner loss of vision in the left eye succeeded.

The report of the oculist was O.D. papillitis; O.S. stasis of the reticular veins. The physical examination gave completely negative results. W.R. of the blood serum negative. Urine normal.

December 5 bilateral clonus of the knee and of the foot set in. Romberg positive. December 7 global paralysis of the left upper extremity. December 8 deviation of the tongue toward the right. December 9 conspicuous dysarthria, dullness of the muscular sense. December 10 marked Brudzinski. Signorelli conspicuous. Babinski positive at the right, doubtful at the left. On the same day very evident paralysis of the inferior facial on the right. The first diagnosis was of tumor of the anterior vermis of the cerebellum compressing the floor of the fourth ventricle at the level of the origin of the root of the accessory nerve of Willis from the bulbus. But the unexpected appearance of the signs of deficiency (supranuclear paralysis) manifested by the right hypoglossal and the right inferior facial roused the question of a mistake. The diagnosis of epidemic encephalitis was discussed. It could not be a case of polyneuritis (absence of pain in the extremities, of paresthesia or of hyperesthesia, of pain on pressure on the nerve trunks, of amyotrophía would go to the establishment of phenomena of indubitable cerebral lesion). It could not even be cerebral syphilis in spite of the W.R. negative except by the absolute absence of phenomena of meningeal irritation through the rapidity and enormous extension of the morbid process. Nor was there thought of sclerosis in plaques because of absolute absence of tremors. The diagnosis of epidemic encephalitis therefore impressed itself. The following facts confirmed our diagnosis. The patient left the hospital, a few days later showed marked sense of euphoria, then restlessness, melancholia, emotivity. The motor functions of the muscles of the face, of the tongue and of the lower limbs returned

and the normal functions became gradually restored so that the patient could distinguish persons and objects placed before her. Much later severe lumbar pains made their appearance accompanied by fever and rectovesicular sphincter phenomena. These unfortunately preceded the patient's death. Since the case constituted a "unicum" so far as regards the amaurosis as an optic neuritis as an initial symptom (in this case lasting about thirty days) I have thought it suitable to publish it. [Author's abstract.]

Nauwelaers, P., and Meunier. LETHARGIC ENCEPHALITIS. [Le Scalpel, March 27, 1920.]

Two cases are here reported from Brussels. The first occurred in December, 1917, just before the epidemic of lethargic encephalitis was recognized in France or England. During the first stage of the disease intense salivation, tremor of the upper limbs, and profuse perspiration were anomalies in the literature at that time. The ocular signs were ill marked, consisting merely of unilateral paresis of accommodation. The peculiarities of the second case were the neuralgia accompanying the onset, slight contracture of the lower limbs, tremors of the upper and lower limbs during the first few days, and the rapid course of the disease, which ended fatally within a fortnight.

Bignami. DUBINI'S CHOREA. [Revist. Ospeali, Nov. 15, 1920, Med. Rec. Ed.]

Within a year a brief history of Dubini's electric chorea in medical literature from an Italian source appeared in the *Medical Record*, in which it was emphasized that this affection was not a mere transient phenomenon, but may be found described in Italian literature as far down as the eighties, covering a period of about forty years from its original description by Dubini. Nothing was said of any later cases. Bignami here reports a case seen by him in 1901 in which the diagnosis was probably Dubini's chorea. This case is given in great detail, including histological studies. Apparently the case was not reported at the time; and in a transcription from the hospital records there was no official diagnosis of chorea or indeed of any affection. But the author now has no hesitancy in making the retrospective diagnosis of myoclonic encephalitis. An exactly analogous case was seen in 1900 in Rome by Sciamanna. These two cases may then be said to represent the transition between the old and new diseases, for the conception of electric chorea had by this time become extinct, while myoclonic encephalitis had not yet been heard from. In January, 1920, Nazari stated before the Academy of Medicine of Rome that the cases of myoclonic encephalitis then prevalent represented a recrudescence of Dubini's chorea. The cases cited above were sporadic, as no massed incidence of such an affection was evident at this time. We note in the case reported that there were no ocular paralyses, and this is true of the historical malady but hardly true of

myoclonic encephalitis of to-day. Apparently this absence may justify the diagnosis of Dubini's disease rather than encephalitis—at least until the significance of this symptom has been finally determined.

Marie, P., and Levy, G. INVOLUNTARY MOVEMENTS FOLLOWING INFLUENZA AND LETHARGIC ENCEPHALITIS. [La Médecine, February, 1920.]

Fifteen cases of various types of involuntary movements following influenza or lethargic encephalitis are here described. The phenomena were unilateral or bilateral, and consisted of choreiform movements, rhythmical contortions, or a fine tremor resembling that of paralysis agitans. Postmortem findings were lesions in the cerebral peduncles, especially in the locus niger, similar to those found in cases of lethargic encephalitis.

De Laroche. FAMILIAL CONTAGION OF LETHARGIC ENCEPHALITIS. [Paris med., Sept. 25, 1920.]

De Laroche states that it is exceptional for the contagious character of lethargic encephalitis to be demonstrated. Out of 114 cases Netter observed only one family in which there were two cases and even then the first case had not been recognized. At Sarrabruck two sisters were attacked, but they were living in an epidemic environment. Laroche reports the case of a gendarme who developed lethargic encephalitis on April 20 in a small town in the Orne, about one and a half months after returning from Lyons, where the disease was prevalent. He was nursed by his wife from the onset of his illness until May 28, when he was taken to the hospital. At the commencement of June the wife contracted a severe form of the disease, which proved fatal within three weeks. As she had not been to Lyons the author considers that she must have been infected by her husband, although she did not develop the disease until forty days after the onset of his illness. Thus the incubation period in both husband and wife was fairly long.

Smith, Wilson. LETHARGIC ENCEPHALITIS. [British Med. Jour., March 8, 1920.]

SIR:—In the leading article of April 24 on "Types and Treatment of Encephalitis" I was interested to see that the use of arsenic and neo-salvarsan is strongly condemned, as I have recently used neo-kharsivan in a case with markedly beneficial results.

On April 3 I was called to see a woman, aged fifty-five, who complained chiefly of intense headache. There was a history of occasional diplopia recently and dizziness; respiration was rapid (55), pulse 65, temperature subnormal. There were frequent involuntary movements of the lips. During the following days lethargy developed; it was with effort that the eyelids were opened, and they very soon closed again. As I had not previously seen a case of lethargic encephalitis, I requested

Dr. J. R. Monro of Spalding, who had some experience, to see the case with me, and he confirmed the diagnosis. On April 9, symptoms having developed steadily, I gave 0.25 gram of neo-kharsivan intravenously. The next day the patient had obviously improved. On April 11 she did not seem so well; I therefore gave a further injection of 0.3 gram intravenously. She felt very ill some three or four hours later, but next day was much better. This improvement lasted several days, but as her condition did not seem so favorable on April 17 I gave 0.3 gram again. This was again followed by a feeling of serious illness a few hours later—not, however, sufficient for me to be sent for—and the next day the patient was cheery and feeling pretty well. She is still doing well.

My object in sending this brief note is to encourage others who might be disposed to try this drug, but who, in face of the very definite pronouncement of Netter, might refrain from its use. I am, etc.

Sicard, J. A. MYOCLONIC ENCEPHALITIS. [*Presse Méd.*, April 14, 1920.]

Five cases of myoclonic encephalitis have come to the author's observation in five months. General lassitude and intense lancinating pains with mild fever occurred in the first week or ten days; then muscular jerkings and twitchings without contracture, spasms, chorea or athetosis followed. During this stage the neuralgic pains subside and there is no somnolency nor ocular symptoms. Toward the third week there may be a tendency to delirium but the reflexes and the pupils keep normal. The course of the syndrome is one to two months. In the terminal stage the delirium is constant but the other symptoms subside, and the patient falls into coma and dies or the delirium subsides and he recovers. It is a milder form of epidemic encephalitis.

Brill, I. C. AUTOSERUM THERAPY IN LETHARGIC ENCEPHALITIS. [*Med. Record*, June, 1920.]

Brill has treated five cases by this method. In four improvement was rapid and definite. In a fifth, the patient was moribund, with signs of developing pneumonia. A single injection of autogenous serum was given intraspinally and the following day the patient's condition appeared to improve, but on the third day he died of pneumonia. The technic consists in obtaining from the patient from 60 to 100 c.c. of blood. A lumbar puncture is performed and 25 to 35 c.c. of spinal fluid withdrawn and an equal amount of patient's blood serum is injected into the subdural space.

Carnot and Gardin. MYOCLONIC EPIDEMIC ENCEPHALITIS. [*Bull. de la Soc. Méd. des Hôp. de Paris*, Jan. 30, 1920. J. A. M. A.]

Carnot and Gardin found lesions in the nerve cells of the cortex and medulla and thrombosis of the veins in the meninges at necropsy of a young man who died the seventeenth day after the onset of ascending paramyoclonus. It started in the legs and spread up to the abdomen, arms and face, with fever and delirium. They also report a case of acute

chorea in a young woman, five months pregnant, with a purpuric eruption and slight fever, with death in less than a week. They do not know how to class these two cases, but incline to label them influenza. Oettinger recalled that during the 1889-1890 epidemic of influenza, Leyden and Guttman reported some cases with narcolepsy (*schlafsucht*) with a fatal outcome in some of the cases. In the discussion that followed, Sainton recalled Dubini's description in 1846 of what he called electric chorea. The description fits some of these cases of epidemic encephalitis. The jerking of the muscles, he said, is like that from an electric current, and it may be accompanied with paralysis or atony of the muscles innervated by the radialis. In Dubini's description of his thirty-six cases of epidemic electric chorea, there is no reference to any other forms characterized by somnolency.

Alexander, M. E., and Allen, H. E. LETHARGIC ENCEPHALITIS. [Archives of Neur. and Psychiatry, May, 1920.]

One of the cases reported by Alexander and Allen terminated in death. Examination of the brain revealed congestion of the meninges with considerable dilatation of the veins. Over the parietal region there appeared, here and there in the pia arachnoid, a small exudate, following the course of sulci. At the base there was intense congestion with a graying yellow exudate of moderate degree in the region of the optic chiasm. Cultures taken from the exudate and from the cortex of the brain showed no growth. Smears showed red blood cells, lymphocytes, mononuclear cells and an occasional polynuclear cell. No organisms were found with methylene blue and acid fast stains. On section of the formaldehyd hardened brain, the ventricles appeared slightly dilated, but the ependyma and choroid plexuses were normal. Sections from the cortex (parietal and occipital lobes) showed in each instance considerable involvement of the meninges. The blood vessels were dilated and there was an accumulation of inflammatory cells in the pia arachnoid consisting chiefly of lymphocytes and mononuclear cells. The cortica gray and subcortical white matter showed dilated vessels, and here and there capillaries surrounded by a few lymphocytes. Sections of the base of the brain showed most marked involvement in the region of the optic thalamus. Here no hemorrhages were evident, but the capillaries were distended with blood. There was marked perivascular infiltration with lymphocytes and mononuclear cells. In places underneath the pia arachnoid and in the substance of the brain an accumulation of lymphoid cells was present. In other places there was a heaping up of endothelial cells of the pia directly in contact with the brain substance. In the pons the vessels were congested, but the perivascular infiltration was less marked. In the medulla, at the floor of the fourth ventricle, the congestion was much more apparent. There were several hemorrhagic areas, the vessels and capillaries were dilated and surrounded by a perivascular infiltration of lymphocytes and mononuclear cells. [J. A. M. A.]

Economo, C. LETHARGIC ENCEPHALITIS. [Polyclinico, March-April, 1920].

Attention is called to this excellent article which is practically a translation into Italian of v. Economo's excellent monograph already reviewed in these pages.

Netter, A. COMMON ORIGIN OF EPIDEMIC HICCOUGH AND OF ENCEPHALITIS LETHARGICA. Successive Chronological Relations of These Manifestations in the Same Subject or in Two Persons in Relation with One Another.

In a series of communications, December 3, 17, 24, 1920, we showed that hiccough had existed in an epidemic state in Paris and its environs and in a certain number of departments since the middle of November. It is difficult not to compare these hiccoughs with the abdominal spasmodic contractions of encephalitis or not to see in them a manifestation of that disease. This frequency of hiccoughs has been noted elsewhere (1919) even before the encephalitis at Paris, in Italy, in Austria, and in Canada. We saw a patient in whom encephalitis was preceded two weeks in advance by hiccoughs. Two brothers living in different localities met at the parents' home December 25 and 26. The elder had been hiccoughing December 21, 22. The younger was taken with encephalitis lethargica December 30. [Author's abstract.]

Gautier, P. EPIDEMIC HICCOUGH. [Rev. Méd. de la Suisse Romende, May, 1920.]

A clinical report of epidemic encephalitis taking the hiccough form in five cases. It came on suddenly, persisting for from two to four days, and then disappearing with equal suddenness, without the treatment having much effect. The patients were four men of thirty to forty-five and one woman of forty-six. The lumbar puncture fluid was found practically normal in the one case examined. Similar epidemics have been reported and the author is disposed to regard them as attenuated types of epidemic encephalitis.

Levaditi, C., and Harvier, P. VIRUS OF EPIDEMIC ENCEPHALITIS. [Bull. de l'Académie de Méd. de Paris, April 20, 1920. J. A. M. A.]

Levaditi and Harvier report the successful inoculation of a rabbit with virus from the cortex, midbrain and medulla of a patient with lethargic encephalitis. The rabbit showed similar lesions in the nervous system and they could be reproduced in other rabbits by inoculation in the sciatic nerve or anterior chamber of the eye. Virus from the clinical case did not prove pathogenic for the monkey and guinea pig until after passage through the rabbit. The virus is filtrable and can be kept in glycerin or desiccated, and can be refound in the spinal cord of animals inoculated in the brain. Convalescents' serum has no neutralizing action on it.

Archambault, L. RARE CLINICAL TYPES OF EPIDEMIC ENCEPHALITIS. [Am. Arch. Neur. and Psych., November, 1920.]

Epidemic encephalitis, aside from the familiar clinical picture of mid-brain involvement, says Archambault in this interesting clinical study, may yield a number of clinical types closely resembling poliomyelitis, paralysis agitans, polyneuritis, cerebellar sclerosis, etc. The combination of choreiform twitchings and acute psychotic disturbance is not uncommon and choreo-athetoid syndromes as sequels of the disorder are frequently obscured.

Maier. ENCEPHALITIS LETHARGICA. [Schweizer mediz. Wochenschrift, March 25, 1920.]

Maier sums up our knowledge of this affection as follows: Up to 1916 we had little definite knowledge of this malady. Its nearest congener is infantile paralysis, which, however, has a pronounced spinal component and affects children by preference. As for any relationship with grippe the more the two are compared the less the suggestion of any kinship, he thinks. Encephalitis lethargica appeared two years before the pandemic. In grippe encephalitis there are no cortical disturbances. Many old practitioners have informed the writer that in the pandemic of 1889-90 there was nothing comparable to sleeping sickness. It is true that patients affected with encephalitis, when placed in wards with influenza cases, did not contract the latter, but this negative evidence is not decisive of anything. The same thing may be said of subjects with other diseases, notably the "false grip" cases. The coexistence of the two maladies in certain localities speaks rather for a common predisposition to both affections, based on lowered resistance. Again, influenza itself lowers the general resistance to disease, so that encephalitis follows in its wake. The new disease is proving very instructive in the demonstration of cerebral physiology, but it is a serious matter, despite its small incidence, with its 30 per cent mortality; and while it comes and goes in the space of three months it can reappear in subsequent waves. Despite the low diffusibility—it is rare for more than one case to occur in a family—it should for the present be considered as mildly contagious, and a certain degree of isolation and quarantine observed. The symptomatology is multifold, and at the onset many conditions may be mimicked as hysteria, dementia precox (catatonic form), and fugues, myoclonus, and chorea. Fever is present in 100 per cent, busy delirium in 78 per cent, lethargy in 82 per cent, muscular rigidity in 78 per cent. Diplopia and strabismus are somewhat less common, but this may be in part explained by defects of observation.

McIntosh, J., and Turnbull, H. M. EXPERIMENTAL TRANSMISSION OF ENCEPHALITIS LETHARGICA TO A MONKEY. [Brit. J. Exper. Path., 1920, 1, 89. Med. Sc.]

This paper describes the apparent reproduction of human encephalitis lethargica in a monkey by inoculation of a filtered emulsion of brain.

Dr. McIntosh, employing a similar technique, had previously inoculated without success into Macaque monkeys and rabbits portions of the central nervous system obtained from more than eight fatal cases. The material for the present experiments was obtained from a girl, aged seventeen years, who died in a virulent, isolated epidemic of encephalitis lethargica in a home for girls at Derby. Dr. MacNalty, who investigated the epidemic, forwarded the brain and brain-stem in 33 per cent glycerin. A summary is given of Dr. MacNalty's description of this most interesting epidemic.

A fortnight after the arrival of the brain, portions of the cervical cord and pons were removed for histological examination. No evidence of inflammation was found in these segments. Portions of the cervical cord, pons, and basal ganglia were washed, and emulsified in sterile saline for inoculation experiments. Of the emulsion, unfiltered, $\frac{3}{4}$ c.c. were inoculated into the cranium and 5 c.c. into the peritoneum of a *Macacus* monkey. On the thirteenth day after inoculation the animal became drowsy and weak. The weakness increased, and on the twentieth day the animal was killed. At necropsy, tuberculous broncho-pneumonia was found. Microscopic sections were made of the mid-brain, pons, and cervical cord. Apart from a few small hemorrhages in the mid-brain, no changes of significance were discovered. Of the emulsion, filtered through a Berkefeld filter, 1 c.c. was inoculated in the cranium and 5 c.c. into the peritoneum of a *Patas* monkey. On the sixth day after inoculation the animal had a fit. From this it had completely recovered on the following day. On the forty-eighth day after inoculation it had a severe fit in which it lay rigid and dazed. From this is recovered somewhat, but considerable rigidity and trembling, associated with lethargy, continued until death on the fifty-sixth day.

Naked eye and microscopic examination revealed no lesions in the viscera. An extensive microscopic examination of the central nervous system is given in detail. In addition to engorgement, hyaline thrombosis, a very few hemorrhages into "perivascular canals," edema of certain cranial nuclei, and extensive degeneration of bodies of neurons, an inflammatory infiltration was found. This infiltration affected, and was most conspicuous in, the posterior part of the left basal ganglia and the junction of the left basal ganglia with the mid-brain; it was also present in focal areas of the cerebral pia and in the wall of a vessel in the right trapezoid body. In its strict localization, its situation, its distribution in the affected area, and in its cytology, it resembled closely the infiltration in human cases.

Other monkeys were inoculated and reinoculated with emulsions of the basal ganglia and cord of this *Patas* monkey. In one reinoculated monkey symptoms have developed. The paper is illustrated by drawings of the infiltrations in the *Patas* monkey and in a human case.

Paterson and Spence. AFTER-EFFECTS OF LETHARGIC ENCEPHALITIS IN CHILDREN. [Lancet, Sept. 3, 1921. Ed. Aust. M. J.]

The name encephalitis lethargica, now generally adopted by the profession in all countries, was first suggested by Economo, an Austrian physician, to describe an epidemic disease which appeared in Vienna in the spring of 1917. A year later a similar epidemic made its appearance in England, but as its prevalence corresponded to a period when tinned foods were a common article of diet, it was at first believed that the disease was a manifestation of botulism with associated ocular and other paralyses. The failure to demonstrate the presence of the *Bacillus botulinus* in the infected persons led to the abandonment of this view and the substitution of another—that the disease was an aberrant form of influenza in which a selective attack was made on the central nervous system. This doctrine was promoted by the fact that a marked increase in the incidence of the disease was simultaneous with the outbreak of the appalling epidemic of influenza which swept over Europe and reached Australasia in 1918. The possibility of a relationship between lethargic encephalitis and influenza is certainly supported by several clinical facts, but the biological evidence in favor of their identity is slight. Another suggestion, strongly advocated by F. G. Cruikshank in England, that the disease was identical with acute infective poliomyelitis (Heine-Medin) but tended to attack the mid-brain and higher centers rather than the spinal cord, has not been supported with any positive evidence. Acute poliomyelitis commonly attacks children, whereas encephalitis lethargica shows no special preference for any age of life. The microscopical hemorrhages which are a common feature of the former disease, are rare and inconspicuous in the latter. Much speculation has been made in regard to these two diseases and the so-called "X disease" described in Australia by J. B. Cleland and A. Breinl. The consensus of opinion is that acute poliomyelitis and lethargic encephalitis are distinct disease processes with no relationship to one another. The relationship of Australian "X disease" to either of these conditions is still a matter of dispute.

Such intense interest has been manifested in the etiology of epidemic encephalitis that the late symptoms and complications of the disease have been largely neglected. Pierre Marie and G. Levy have pointed out that although generalized choreiform movements disappear usually at the end of two or three months, localized rhythmic movements and muscular weakness may persist for a year or more. The tremors and rigidity of the paralysis agitans type of the disease may persist for an indefinite period. Little attention, however, has been given to the residual mental symptoms, although the lethargy, irritability, hallucinations and transitory delusions of the disease when at its height have been thoroughly studied.

In a report made to the Local Government Board in 1918 A. S. Macnalty pleaded for caution in connection with the advancing of any

opinion in regard to the after-effects of the disease. He believed it possible that mental changes and residual paralyses might prove to be not uncommon sequelæ. About the same time E. Farquhar Buzzard stated his conviction that epilepsy, mental deficiency, hemiplegia and diplegia would prove to be permanent results of encephalitis occurring in childhood.

Quite recently Donald Paterson and J. C. Spence have attempted to inquire into the truth of Farquhar Buzzard's prediction, on which study this paper is founded. They selected twenty-five children between the ages of three months and eleven years who were without doubt suffering from the disease in a typical form. Three of the four children described by Batten and Still in a paper on "Epidemic Stupor" published in 1918 were included in this series. Batten and Still made no reference to any after-effects exhibited by these patients. Paterson and Spence found that one of the three was now an inmate of a mental hospital. She showed marked mental deficiency and failed to recognize even her own parents. The second child was dull and slow to learn, lacking the normal child's interest and buoyancy. The third, a boy aged fourteen years, who had been a normal, intelligent boy prior to his suffering from encephalitis lethargica in 1918, from which he appeared to make a complete recovery, has become a criminal since his illness. Theft is his besetting sin and the police already regard him as an habitual criminal. Of the authors' twenty-five patients, only one died. Of the remaining twenty-four, only six have completely recovered from their illnesses, no physical or mental trace of which remains. Eighteen show various degrees of mental deficiency. Seven of these show all the mental stigmata of pronounced and permanent idiocy. They are woefully lacking in intelligence, show no recognition of their parents and certainly no affection towards them, drool from the lips and make the quaint grimaces and gestures characteristic of the congenital idiot. The remaining eleven show slighter degrees of mental deficiency—all dating from the illness from which they suffered. Dull and backward, slow to learn, with just sufficient intelligence to avoid danger in the streets, some of them show a moral obliquity which augurs badly for their future. Those whose habits cannot be described as actually criminal, are mischievous and untrustworthy, causing added anxiety to the mother, who finds it difficult enough to control the antics of the average small boy.

But the disastrous effects of the disease are observed in the physical as well as in the mental condition of the children. The children are well nourished, but are slow to develop the characteristics of normal children. Some of them, for example, reach the age of two years before they are able to sit up. The power of standing or walking is likewise delayed. Paterson and Spence insist that the slow physical development is a result of the mental deficiency and is not due to organic palsies. Seven of the eighteen affected children are still suffering from organic residual paralyses resulting from the disease. Two suffer from typical spastic

diplegia, one from hemiplegia with contractures, three from ataxia and incoördination of the arms and one from so-called Parkinsonian syndrome.

The authors' paper reads like an appalling commentary on the after-effects of the disease. If their main observations are correct (and they certainly appear to have been carefully made), encephalitis lethargica has more terrors than the immediate risk of death. Perhaps Paterson and his colleague happened upon an unusually bad series of children. Further observations and the experiences of other physicians will be anxiously awaited. Nothing concerns a nation more than the welfare of its children.

Barré and Reys. LABYRINTHINE FORM OF EPIDEMIC ENCEPHALITIS. [Paris méd., October, 1921].

During the recent epidemic of encephalitis they were struck by the large number of patients who complained of vertigo, loss of equilibrium, and lateropulsion, these symptoms appearing earlier and being more pronounced than the ocular symptoms or drowsiness. The frequency of these cases induced the writers to describe a labyrinthine form of epidemic encephalitis which might occur in association with other signs of encephalitis or in a pure form. Examples of the first group are much the most frequent, those of the second group forming only 12 per cent of the cases observed by the writers. The pure form develops in the following way: The patient, who has previously been perfectly healthy, suddenly develops attacks of giddiness which last only a short time but return at more or less frequent intervals. At the same time the patient feels drawn in a certain direction, or walks like a drunken man. He feels extremely weak, and the sensation of heaviness in the head prevents him from undertaking any work which requires sustained attention. The symptoms are relieved by rest and silence and are aggravated by sudden movements or noise. During the attacks of vertigo the patient may sometimes see double. The condition is liable to be mistaken for neurasthenia, hysteria, cerebral arteriosclerosis and softening, Ménière's disease, disseminated sclerosis, and subacute labyrinthitis due to other infections than epidemic encephalitis. The diagnosis from hysteria and neurasthenia is made by the presence of nystagmus, Romberg's sign, and the Babinski-Weil test. Treatment consists in administration of quinine in doses of 10 or even 5 cg. twice or thrice a day, a considerable time after food. Several patients have also derived benefit from cachets containing stovaine 0.01 gram, pyramidon 0.20 gram, and veronal 0.25 gram.

Denyer, S. E., and Morley, D. E. MYOCLONIC ENCEPHALITIS SIMULATING RABIES. [Br. M. J., February 5, 1921.]

F. G. Crookshank in 1918 recalled to mind old citations of "hydrophobia without dog bite," and the relationship to influenzal encephalitis,

and it is in the ken of the abstractor of some internists who have insisted on the psychogenic nature of true hydrophobia. These authors show how a myoclonic encephalitis can present the picture of a hydrophobia.

Kling, Davide, and Liljenquist. EXPERIMENTAL EPIDEMIC ENCEPHALITIS IN RABBITS. [*Hygica*, November 16, 1921. B. M. J.]

These authors make a further report on their experimental work in inducing typical epidemic encephalitis in rabbits by the intracerebral injection of material obtained from the brains, throats, and feces of patients who had died or were suffering from typical epidemic encephalitis. In a few instances the rabbits died in a week or two after inoculation, but as a rule the disease lasted several weeks or months, and one of the rabbits did not die till seven months, after inoculation. Even when rabbits were killed during the incubation period, and before the appearance of definite cerebral symptoms, marked changes in the brain, characteristic of typical epidemic encephalitis, were sometimes found. The authors suggest that the same events may occur in man, the disease existing in the brain but running an abortive and more or less symptom free course. Like syphilis, epidemic encephalitis appears to run a capricious and, at times, seemingly inactive course in the central nervous system, and the duration of the disease may be one of weeks, months, to be invisible, refractory to culture, and glycerin resistant, and even years. The authors found the virus to pass a Berkefeld filter,

Orr, D., and Sturrock, A. C. LOCALIZATION OF TOXI-INFECTIVE LESIONS IN THE CENTRAL NERVOUS SYSTEM. [*Br. Med. J.*, June 3, 1922.]

At a meeting of the Manchester Pathological Society these authors read a paper entitled "The influence of disturbance of the sympathetic mechanism on the localization of toxi-infective lesions in the central nervous system." They stated that lymphogenous toxi-infections of the central nervous system followed definite nerve root paths and were primarily systemic. Nonsystemic lesions such as those of pernicious anemia, Addison's disease, and cancer cachexia were the result of blood-borne infection, but to call them hematogenous did not adequately explain their distribution. Degenerative changes in the nonsystemic lesions or combined scleroses in the cord were found over the surface of the white columns and around the postero-median septum, which parts derived their blood supply from the pia-arachnoid, whilst the grey matter and most of the basis bundles, which were supplied from the anterior spinal artery, escaped. The stress of these lesions fell on those segments of the cord from which white sympathetic rami passed. In the brain the same distribution was found, lesions in a general blood infection being found in parts supplied by pia-arachnoid.

Experimental work was carried out on rabbits. First, a study was made of vascular changes in the brain after division of the cervical sympathetic, and secondly of vascular and nervous elements in the brain in

similar animals after flooding the circulation with an emulsion of B. dysenteriae Shiga injected into the vein of the auricle. The results might be summarized as follows:

Lesions due to sympathetic division alone. These were vascular dilatation, perivascular edema, slight proliferation of adventitial cells which might be a response to increased permeability of the vessel wall resulting from sympathetic paresis and the release of certain noxious products from the circulation. There was at the same time some morbid change in nerve cells, and the areas affected were those supplied by pial vessels—namely, the cortex cerebri and the cornu ammonis, the fascia dentata, the amygdaloid and caudate nuclei, which were supplied by folia of pia carrying vessels invaginated from the surface of the brain.

When, after division of the cervical sympathetic, a general intoxication was produced, lesions were found affecting the same areas, but to a much greater degree, and the unilateral sympathetic division intensified the morbid lesion on the same side. The areas involved in both series of experiments were the archaic regions of the brain *plus* the cerebral cortex, which was developed from the archipallium. All were supplied by pial vessels and under sympathetic control. It would appear, therefore, that the sympathetic nervous system played an important part in toxic-infective inflammation and degeneration and its involvement was a contributory factor in the localization of lesions, not only in the central nervous system, but probably wherever they might occur. Further investigation on this point might lead to a better comprehension of the genesis of obscure nonsystemic lesions affecting both spinal cord and brain.

While lesions were found in all the areas mentioned above, the most striking and remarkable was extensive periarteritis in the head of the caudate nucleus just external to the anterior horn of the lateral ventricle. This lesion was so well defined and so localized as to seem of special significance in view of the obscurity which at present surrounded the etiology of certain lesions which implicated the lenticular area.

In these experiments on acute intoxications ependymal cells of the lateral and third ventricles and of the iter, as well as the epithelium of the choroid plexus, were in a state of active secretion. This secretion consisted of small globules of a translucent and highly refractile material, which coalesced into larger masses, consisting of an inner core with a weak affinity for certain stains and an outer marginal zone with a marked affinity. This was an active secretory process of a lipid material, which was probably of the greatest importance as an active defensive measure against intoxication as well as one destined for the purposes of regeneration and repair.

These lesions occurring in the archaic part of the brain and intensified by disturbance of the sympathetic mechanism as well as in the superficial layers of the cortex cerebri, might play an important part in the genesis of disturbed emotional states. They were produced through interference

with the blood supply without added toxin, but were greatly increased by the additional factor of an experimental intoxication. This fact inclined the authors to take a much broader view of the genesis of emotional disturbance, and tended to take the question out of the purely psychical field of argument. In emotional disturbance the initial stimulus might come from the sensorial cortical areas, the peripheral sensory nerves of the life of relation, or through the endocrino-sympathetic system, which subserved the function of kinesthesia and was the physiological foundation for those feelings of well or ill being so important in the psychic and physical life of the organism. These kinesthetic or vegetative stimuli, when normal, remained subconscious, but leapt into prominence so soon as they became abnormal. Every stimulus which impinged on the central nervous system produced results which embraced a vast territory, and whether the exciting stimulus be central or peripheral its influence reverberated throughout the whole nervous system and its appendages, and was certain to find its expression in a reaction on the part of the psychic and vegetative life.

A healthy affective tone played no small part in keeping the higher cortical and intellectual functions up to their highest level, and affective tone and emotion were influenced or often greatly disturbed by morbid changes in the archaic portions of the brain. It was there and in the cerebral cortex that lesions occurred after interference with the cervical sympathetic in the rabbit, and as affective tone and emotion were so closely linked up with the function of the endocrino-sympathetic system their disorder was probably the result of a psycho-physical process which altered the nutrition of the ganglion cells which governed their mechanism. A similar process was no doubt accountable for the large group of functional disturbances termed the "psycho-neuroses." Future researches might ascribe more importance to the effects of altered nutrition of the central nervous system than to the purely psychic element, though one recognized the influence of the one upon the other.

Da Fano, C. HISTOPATHOLOGY OF EPIDEMIC ENCEPHALITIS. [B. M. J., Jan. 29, 1921.]

C. Da Fano states that in the nervous tissue from cases of epidemic (lethargic) encephalitis within and without the nerve cells minute forms have been observed, to all appearance consisting of a central generally basophil particle and of a delicate little stainable body, irregularly round or oval in shape. For these forms the term "minute bodies" is *pro tempore* proposed. The bodies are generally discrete, and provided with one granule, but dumbbell-shaped forms occur as well as others with two central particles arranged in pairs. An as yet not quite definable relation seems to exist between these forms and a granular, pigment-like material occurring within the nerve cells in places where brown or black pigment is not generally found. Minute forms, similar in shape, structure, and staining properties to those observed in the nervous tissue, have been

traced within and without the cells infiltrating a salivary gland from an acute case of the disease. After discussing the supposition that such findings might be the product of an optical illusion, associated perhaps with degenerative changes caused by the malady, the suggestion is tentatively put forward that they might be due to the presence in the tissues of a living agent, cause of the disease.

5. CEREBELLUM.

Stenvers. DIAGNOSIS OF CEREBELLOPONTINE TUMORS. [Ned. Tijds. v. Genees., November 6, 1920, II, No. 19. J. A. M. A.]

Stenvers states that in five of the six tumors of this kind he has had to examine, the neurologists and ophthalmologists consulted had diagnosed the case incorrectly until the roentgen-ray findings in the petrous bone gave the clue. He describes further a case of trigeminal and occipital neuralgia, with facial paresis, right hypoglossus paresis, slight changes in the fundus of the eye, abnormal corneal reflex, slight nystagmus, a tumor in the vicinity of the ear, disturbance in taste and partial deafness, with extreme sensibility to loud sounds. Muskens has reported an almost identical case and explained it as a cerebellopontine tumor, but in Stenvers' case necropsy revealed that the causal lesion was an extracranial metastatic tumor protruding into the middle cranial fossa. The pontine angle was normal. He adds that in every case of cerebellopontine angle tumor which he has examined, the petrous portion of the temporal bone was always found decidedly abnormal, and the roentgen examination invariably had revealed this anomaly in the petrous bone during life. The clinical-neurologic diagnosis should always be confirmed by roentgen examination of the petrous bone (*rotsbeen*).

Vail, Harris H. TUMORS OF THE NERVUS ACUSTICUS. [Laryngoscope, August 1920.]

The findings in ten cases of verified tumor of the eighth nerve by the Bárány rotation and caloric tests are here reported upon. Typical reactions were obtained in the majority of the cases. (1) Unilateral deafness varying from absolute to a marked involvement on the side of the lesion. (2) Failure to obtain after-nystagmus and past-pointing from stimulation of the labyrinth on the side of the lesion by the caloric test, and in some cases failure to obtain after-nystagmus and past-pointing from stimulation of the vertical canals on the side opposite to the lesion by the same test. (3) By rotation the time of the after-nystagmus as a general rule is decreased, but does not show that one labyrinth is blocked. Rarely there is found a halving of the after-nystagmus time when rotation to the side opposite to the lesion is done. This is due to the compensation resulting from the chronicity of the lesion according to the author. (4) Following rotation, as a rule, past-pointing tests do not show reactions that may be classed as classical. The reactions are irregular and are influenced by direct or indirect involvement of the cerebellum, chiefly

from pressure. (5) In two cases with failure to produce nystagmus by the caloric test of the unaffected side, when similar tests were made after operation, they showed absence of nystagmus and past-pointing reactions on the side of the lesion, with practically normal response from stimulation of the opposite side. The author thinks that the side opposite to the lesion was affected by increased pressure and that this caused to a great extent the irregular reactions found by the rotation test. (6) Post-operative tests of the cochlea on the side involved showed an increase in its function in some of the cases. (7) Unilateral paralysis of the external rectus muscle may cause unequal involvement of the eyes in spontaneous nystagmus. In after-nystagmus by stimulation this is a negligible factor. (8) The reactions showed that often, as in lesions of the cerebello-pontine angle, the vertical semi-circular canals of the side opposite to the lesion fail to respond to stimulation.

Hausmann. CYSTICERCUS IN CEREBELLUM. [Schweiz. med. Woch., December 2, 1920, L. No. 49. J. A. M. A.]

The young woman was suddenly affected with vertigo and vomited; poisoning from a dish of sauerkraut was assumed, as the mother had vomited at about the same time. But the mother rapidly recovered while the symptoms in the young woman continued a progressive course, imposing the assumption of a tumor in the right side of the cerebellum. While an operation was being considered the patient died, and at necropsy a cyst was found at the anticipated site. It shelled out readily and proved to be the work of *Cysticercus tenuicollis*. The right lobule of the cerebellum had been pressed into the foramen magnum, which explained the peculiar way in which the patient had held her head bent over to the left.

BOOK REVIEWS

Towner, R. H. *THE PHILOSOPHY OF CIVILIZATION.* In Two Volumes. [G. P. Putnam's Sons, New York and London, The Knickerbocker Press, 1923.]

The Preface of this book gives promise that one will find in its content much food for serious thought toward the redirection of forces now disastrously at work in civilized society. The second volume in large part bears out this promise. The first does not. Various suggestions tending toward a truer apprehension of facts are buried beneath the author's mass of material utilized with a marked weakness of logic. One is confronted by this material gathered from other men's histories and philosophies of civilization, a collection that speaks for the author's erudition but not his intellectually selective and assimilative powers. He quotes enormously but with an uncritical acceptance of these large portions of the writings of others, he takes ancient biblical legends very literally and all this to uphold the thesis which he sets forth. He uses this like a machine set to reduce everything to its prescribed formula, to which he ascribes mathematical certainty, proved in each instance by the material freshly brought forward by him.

This thesis is that civilizations rise and maintain themselves so long as the group is replenished from sexually frigid mothers, who become mothers only from a sense of duty and transmit their sexual coldness to their offspring. Their sexual aversion tends toward a higher nervous organization and the group is constantly improved. On the other hand, as soon as a false religious ideal permits these women to find other duties than motherhood, or any other factor leaves this function to women of such a low order that they enjoy the sexual relation, the trouble begins and civilizations are on the decline. It matters not in either case how lusty the father may be. Thus the author manipulates to his liking the laws of heredity and of psychology or rather, for the greater part, they seem to be disregarded.

In the second volume the author treads on firmer ground in his observance and appraisal of the tendencies of to-day repeated from earlier civilizations. Yet here this thesis of his is dragged in repeatedly not as if it had an indispensable place in his later argument but as if an earlier loyalty to it must not be entirely forgotten. It mars rather than aids his more practical consideration of the matters under discussion. Even in the treatment of these, excellent as are his suggestions, there are present other faults of the first volume. His theory of the place of alcohol in civilization, while containing much of historical and psychological weight, is one to which the most de-

voted adherent of the use of alcohol would find it difficult completely to subscribe. He sets forth much of serious fact in regard to the dangers of uniformity forced upon mankind to destroy the more vital diversity, he emphasizes the subtle evils lurking in espionage and authority. His discussion of the relation of intellect and emotion and the chapters in which he points out the benefits of a consciously directed civilization as against a fortuitous one are interesting and thought provocative. Yet in it all he writes as a man not himself moving clearly through facts and their implications. His psychology is not broad and plastic enough to carry his vision where it strives to go. His closing pages in which he advocates for the saving of civilization the following of one man's gospel, which, he says, "is a sure guide to the benefits of mathematical law, and its promise . . . universal," confirms the feeling otherwise gathered through the reading of the book that the author himself is still hampered by authority in thought and ideal.

Ruggles, Gates R. HEREDITY AND EUGENICS. [The Macmillan Company, New York.]

Of the many recent contributions to the general discussion of heredity this extremely well written book can be recommended. It contains an admirable series of descriptive definitions of the Mendelian concept of heredity, some profitable discussion on the inheritance of physical structures, a less carefully considered chapter on the relationship of mental traits to inheritance with much debatable material, debatable because not carried deep enough into the background of psychology, where the general Adlerian point of view and Semon's Mneme theories might have been presented to the advance of both the geneticist and the analytical behaviorist in psychology.

There are then added some fascinating even if glittering chapters on Eugenics. It is an excellent performance and a very readable book.

Holmes, S. J. STUDIES IN EVOLUTION AND EUGENICS. [Harcourt, Brace and Company, New York, 1923.]

This book presents a series of brief chapters, direct in the manner of setting forth their subject matter and concisely formulated to bring this matter unavoidably to the reader's thoughtful attention. The earlier chapters are devoted to a review of the history of the theories of evolution since Darwin's day. The author's fairness and breadth of objectivity are in evidence here. He is able, with such a point of view and because he is at home in the field of biology, to view the various theories as flexible and alterable means for gaining further knowledge of life and for exercising control upon the processes of life.

In this latter interest he presents chapters of serious practical importance. In the same spirit which pervades his more theoretical dis-

cussions he examines some of the pertinent questions of the day upon their biological background. He is not inclined to take the darkest view of these matters. He inclines rather toward an optimism which he almost confesses openly is born of wish. His supporting facts are not of the amplest, therefore his conclusions may be questioned at times. Yet his book is not a record here of dogmatic conclusions but an attempt to look upon both sides of important sociobiological questions and to find out as broadly as possible whither the factors involved are tending. Eugenics, birth control, immigration and the present status and future of the negro race are among the topics which pass under review. His manner of treatment does much to clear any of these from the misconceptions which surround them and from the ill-considered opposition or support which might be accorded any of these matters on the basis of such misconceptions.

Kroeber, A. L. ANTHROPOLOGY. [Harcourt, Brace and Company, New York.]

We would like to write a long, critical review of this interesting work, but we have not enough space and our readers are not primarily anthropologists although as students of behavior, either at metabolic or at social conduct levels, the foundations which anthropology deals with are constantly showing through.

We do want to say, however, that we find this one of the most interesting and satisfactory presentations of anthropology which has appeared since Tylor's older classic. Our readers will find it very much worth while.

Siemens, H. W. EINFÜHRUNG IN DIE ALLGEMEINE UND SPEZIELLE VERERBUNGSPATHOLOGIE DES MENSCHEN. [Zweite Auflage. Julius Springer, Berlin.]

The first edition of Siemens' book gave promise of a very practical series of condensations relative to the rapidly expanding issues connected with Mendelism. It was too short and didactic and failed to be satisfying.

The present edition, appearing two years later however, has brought the discussion of the problems to a most gratifying status and no student of genetics can fail to get most valuable guidance from this present rewriting.

In spite of the fact that so far as neuropsychiatry is concerned the hereditary mode of approach leads us much more into difficulties than into the clearance of our problems, there are nevertheless many pertinent suggestions which should lead to the greater definition of many nosological conceptions. Thus Harnhart has seemed to show that there is a type of Friedreich's ataxia with a comparatively pure Mendelian modus of inheritance. This should help us to arrive at certain fundamental pathological analyses of the systems involved. If defect states interpretable in terms of the phylogeny of nervous elements are recognizable then we have come a long way in the setting apart of definite syndromes on an hereditary basis.

In this as in other disease groups this work will be of service in sharpening our insight and in raising the demand for more stringent pathological analyses.

Pfeifer, R. A. DAS MENSCHLICHE GEHIRN. 4 bis. 8 Auflage. [Wilhelm Engelmann, Leipzig.]

We have favorably reviewed the third edition of this small work on the human brain which appeared in 1920. A new edition, somewhat enlarged, here is offered. The value of this presentation is its concise statement and apt illustration of the chief functions and structures of the human brain. The author is thoroughly related to his subject, and although little is new, and only a sketch is offered, what is told, is told from a broad general viewpoint.

Yellowlees, Henry. A MANUAL OF PSYCHOTHERAPY. [A. C. Blach, Ltd., London.]

If the author had entitled this a primer of some psychoanalytic principles with notes on suggestion, autosuggestion and hypnotism, such would have been a better descriptive title than A Manual of Psychotherapy, for psychotherapy runs the gamut from "goose stuffing" to the most atechanical scientific principles of the analytic philosophy. Of this range the author gives no real hint, but deals with only the more in vogue analytic situations.

In this narrower sense then we entertain a high opinion of this small manual since it goes to the heart of the more important of the problems involved. It is a very excellent, clear and common sense discussion of the more readily comprehended psychoanalytic principles with some short descriptive chapters on suggestion, autosuggestion and hypnotism. It will prove a useful book.

Brierley, Susan S. AN INTRODUCTION TO PSYCHOLOGY. [Dodd, Mead and Company, New York, 1923.]

This volume merits for itself a worthy place among the books on psychology which abound to-day. It comes like a clean-cutting pathfinder where many writers are wandering from the road or pursuing laboriously roundabout routes. The book is what its author promises it shall be, a guide to the beginner or other worker without a special training in psychology. It presents to such—or to the already instructed reader—a clarified statement of the science of psychology and its application all along the way to the understanding of the working of the human mind even to the investigation into its deeper content. The author makes no boast of an exhaustive study of all this field but her presentation gives the necessary preliminaries for further study at the same time that it reveals the proper value and relative significance of the elementary facts. They are well defined while the conception of these facts as parts of the genetic development of human experience prevents any undue emphasis of any particular phase of psychological experience or any too formal ad-

herence to only one method of approach to psychological fact. The matters of unconscious psychology are treated with the same direct acceptance as the longer accepted matters of conscious psychology. The author seems to be able to admit the forum as neither separate nor hostile to the science of psychology but as part of its legitimate field. She is capable of stating briefly the Freudian principles with a comprehension of their actual significance and their place in the illumination of the knowledge of the human mind which many of his critics might well make their own. She makes the transition from the revelations of the psychology of consciousness to those of the unconscious appear as a most natural sequence.

Russell, David. THE EVOLUTION OF CONTINUITY IN THE NATURAL WORLD. [Moffat, Yard and Company, New York, 1923.]

This is a book of intense interest, clear, direct in its presentation. It is in every way suited to win the reader's attention to its original view of evolution and to its insistence upon the fact of the sudden acquisition of variations. "*Natura non facit saltum*" is a fallacy, according to the author's point of view, and he delights in refuting it. He views evolution in the individual form or in the larger group, the evolution of matter and the appearance of life within matter as evidences of one and the same activity of force. The double law of attraction and repulsion, the former gaining ground, brings about an alternating continuity and discontinuity. All growth is evolution and this may be expressed for the higher forms of the living individual by saying that its cycle begins by the attraction of separate elements toward continuity and ends with the restoration again of the separate elements, the sexual elements, through discontinuity. This is the principle found at work everywhere, in the atom, in the molecule, in the plasmolecules, as the author is pleased to denote the component elements of living protoplasm, as well as in the process of cell multiplication and differentiation. The ascendancy of attraction has made for continuity of a part of the Individual even while discontinuity has permitted the return to the elements which reinitiate the cycle. Environment has acted upon the simpler forms of this continuity to intensify the latter to higher forms. Control and arrest are auxiliary factors which have been developed along with the increase in continuity. Russell traces this simple process through various evolving forms setting forth with particular detail this "evolution of continuity" in the production of invertebrates and vertebrates accounting for their similarities and differences in development. He emphasizes through them what he believes to be the truth in the evolution of forms, that all terrestrial forms represent only the advanced ends of evolutionary side types. The straight road of evolution which has culminated in man has left no traces of itself in any terrestrial forms. For the straight road was formed in an aqueous environment. The segmentation which produced the vertebrate as well as that which developed the invertebrate was already evolved before a terrestrial habitat was found. Water pressure has been a

large part of the environmental means through which repulsion and attraction have worked to produce the intensified continuity which resulted in advancing forms. The action of this environment upon the developing form at a particularly effective time of its development came about suddenly so that every evolutionary variation has been a sudden acquisition, however gradually it may have become manifest.

The book deserves careful reading in order to pursue the argument by which the author's theory is so interestingly developed and his application of it to all forms of the evolution of matter, living and nonliving. The principle set forth not only is a fruitful one for biological investigation but it constantly suggests most stimulating psychological parallels.

Micklem, E. R. *MIRACLES AND THE NEW PSYCHOLOGY. A STUDY IN THE HEALING MIRACLES OF THE NEW TESTAMENT.* [Oxford University Press, London, Humphrey Milford, 1922.]

This book makes a valuable contribution along the way which the writer suggests as one to be pursued in the future. He applies the principles of the "new psychology," as these are revealed in the various forms of psychotherapy at the present time, to the possible explanation of the apparently miraculous healings of the New Testament. The book forms, therefore, an unprejudiced review of these various methods as illustrative of present day psychology and it presents a simple yet scholarly study of these recorded phenomena of the past. The historicity and the theological significance of the New Testament records is skillfully touched to be set aside as the background only relatively essential to the psychological significance of the conceptions of disease and the matter of their healing. The writer has opened a field where further application of the psychological principles would bring still clearer understanding even of the significance of the efficacy of faith in a conception of God, upon which he lays final stress. Interesting comparison is made between disease and cure as pictured in the biblical narrative and similar conceptions and experiences in the modern world, the more enlightened and the more openly superstitious portions of mankind.

Mensendieck, Bess M. *FUNKTIONELLES FRAUENNATUREN.* Mit 164 Abbildungen nach Naturaufnahmen. [F. Bruckman, A.-G., München, 1923.]

The author enters an earnest plea for the complete functional control of the human body, the woman's particularly, through the taking thought upon it by each individual possessor of such a body. She contrasts such a method of understanding of the body and of practiced control of it with that of the gymnasium, sport, or the dancing school. Either of these fails to enter completely into the daily use of the human body. Naturally the sway of each is limited and the first particularly has also the disadvantage of submission to another's thought rather than the intelligent functional control which belongs to Mensendieck's ideal. This looks deeper into the meaning

of the body with its unlimited capacities for freedom in purposeful movement and posture. Mensendieck decries our boasted automatic efficiency, comparing it with the more graceful and more effective movement to be seen in the child or the animal, pertinently asking whether the animal's movements are not something else than automatic. She has set forth in this little book, in addition to these instructive principles, a practical guide to the knowledge of the capacities of the human body with abundant illustrations for self training in discovering them. She lays emphasis upon woman's responsibility for the better appreciation and training of the body. It must be the object of thought and of unobstructed observation, too, and this must be instilled into the children. Such training, mental and physical, can only make for true appreciation and act as a corrective to the following of empty dictates of fashion or meaningless "expressionism" when there is nothing to express.

Satow, Louis. HYPNOTISM AND SUGGESTION. Translated by Bernard Miall. [Dodd, Mead and Company, New York, 1923.]

This book is brilliantly written. It presents in a manner suitable for general information a survey of the practice of hypnosis and the psychological principles upon which it rests. The work is written with the intention "that he who runs may read" but the runner will be compelled to halt and think. It is not a popular book in the sense of easy superficial reading. Moreover, it should stimulate the reader to stop for self examination for it is a treatise upon the slavery of humanity everywhere and at every age, ours no less than those past, to superstition and obedience to authority instead of clear interrogatory thinking. There is, therefore, a blind infatuation in regard to the phenomena of hypnosis and suggestibility as to the mysteries of a cult. These phenomena the author reduces to simple psychological facts. He explains how they arise and how they may be utilized therapeutically or how they have been employed for good or for evil when not scientifically understood. This is all told in vigorously direct language which enlists and holds the reader's attention. The evils wrought in our own recent years by the working of the phenomena discussed in the movements of "mass psychology," leading to the maintenance of a belief in war, to precipitation into war and to other disasters to mankind, become a pertinent part of the presentation.

We do not feel satisfied with Satow's definition of hypnosis and suggestibility. He states a preference for the word "unconscious" but then continues to use the term "sub-conscious." Perhaps a stronger feeling for the former would have swayed him had he gone further into the fruitful ground of the unconscious for the reasons why hypnosis may be induced and suggestion prove effective. Would he not have found here tendencies which form the final explanation for these things which, with all his clearing of the debris of mystery which surrounds the phenomena, he still leaves too much as if induced from without. The absence of any reference to psychoanalysis and

those who have approached these problems through it seems almost a studied avoidance. It constitutes at least an historical omission noticeable among the writer's otherwise wide range of references. The translator, for some reason, has seen fit to append a borrowed psychoanalytic glossary, which he duly acknowledges. Perhaps he felt the defect in this otherwise excellent book. We are indebted to the translator for making the work available in English.

Bellwald, A. M. CHRISTIAN SCIENCE AND THE CATHOLIC FAITH. INCLUDING A BRIEF ACCOUNT OF NEW THOUGHT AND OTHER MODERN MENTAL HEALING MOVEMENTS. [The Macmillan Company, New York, 1922.]

This study forms a document of no little human interest. It presents the mind at work upon the problem of its own relation to the body as regards health and disease at many different periods and in many different fashions. It shows, too, the close relation of these varying points of view. Occupied chiefly with Christian Science as its main topic it reveals much of the nature of the founder of the latter. Her overwhelmingly egoistic nature overrode obstacles to build its own peculiar success and establish a system capable through its very crudities of a subtle enticement of large numbers of men and women. The writer leaves much of the deeper psychology of all this still unexplained. Yet his thought as far as it goes—he has not pursued the psychology of the unconscious—reveals a broad background of knowledge of the history of thought and of the psychology of human life. Against this his exposition plays so lucidly that the book becomes a valuable historical contribution in regard to Christian Science and other modes of mental healing and also as concerns the writer's own place as a Catholic thinker. Here his position is somewhat dogmatically restricted. He has failed to advance understandingly into some of the things which he too readily dismisses. For example, whether he counts Freud "recently defunct" personally or only in regard to his teachings, either is an inaccuracy from which he should have saved himself. Yet the sympathetic breadth of his attitude is not seriously impaired. The book is still stimulating in its presentation of the matter contained and of vital quality through the author's own thought.

Laird, Donald A. AN INTROCUCTION TO APPLIED PSYCHOLOGY FOR NURSES. Illustrated. [J. B. Lippincott Company, Philadelphia and London.]

Laird presents psychology to the nursing profession as something very practical and very human. He gives his presentation of it a sound basis in neurology and behind this in biology. For he shows that not even the nervous system furnishes the whole physical structure of the psychic life. The latter is a matter of adaptation of the organism more fundamental and farther reaching than the extent of the nervous system which it has specially prepared for itself. The endocrinous glands therefore come under consideration as well as the most primitive physicochemical responses of the cell.

The author thus stimulates the nurse to regard herself or her patient as a fundamentally vital reactive organism. The more complex processes which appear as human psychic behavior are considered upon this ground with regard also to the disturbances which make themselves observed. Attention is given to the mechanisms by which they arise and by which they get control of the personality. Thus very briefly though suggestively the nurse is led to understand marked or less obvious aberrations in mood and behavior but far more than that she is taught to observe the psychic reactions proceeding daily in herself and those with whom she has to do upon the background of their meaning and their mode of action. Stimulating topics for further thought are appended to each chapter. The meaning of psychic reactions is all too briefly touched. The writer skillfully points to an unconscious content and discusses briefly some of the material of it. But even in a volume of this scope something more of its importance in mental disturbance as well as in those daily manifestations toward which the nurse must direct her attention should not be left unspoken.

Hesnard, A. *L'INCONSCIENT*. Bibliothèque de Psychologie Expérimentale. Directeur Dr. Toulouse. [Libraire Octave Doin, Paris; Gaston Doin, Editeur, 1923.]

This study of Hesnard gives evidence of the naturalness and inevitableness of the recognition of the unconscious in a really comprehensive psychology. Hesnard surveys the many forms in which the fact of an unconscious realm to the mental life makes itself felt as he brings evidence also of the content of this part of the psyche and its dynamic activity. He is careful also to insist that this mental activity is not to be considered apart from the entire life of the organism, physical as well as psychical. Nor from the more strictly psychological point of view are consciousness and the unconscious to be considered apart but there is "one and the same activity—conscious or unconscious according to the circumstances." Hesnard states in psychological terms the development of the unconscious as it comes to us by heredity in its ontogenetic aspect or viewed phylogenetically. He discusses its nature which is affective rather than rational and presents a study of its relation to receptivity and as a source of motor activity touching here of necessity upon its relation to consciousness. This brings him inevitably to its importance in affective disturbances in the milder neuroses or in the psychoses. It includes also a survey of the unconscious as the reservoir from which arise those phenomena which are often ascribed to supra-normal sources. Hesnard finds them sufficiently accounted for by the acceptance of the unconscious activity beneath the superficial activity of consciousness. Among the methods of approach to the problems of the unconscious as related to medicine, briefly reviewed, Freud's position is given consideration and frequent reference is made to it although the author is not in full agreement with him. The book is valuable as a clear and comprehensive survey of the unconscious as a subject no longer to be ignored in psychological literature. It contains a very full bibliography.

OBITUARY

DR. HAROLD N. MOYER

Dr. Moyer was born August 14, 1858, and died December 14, 1923. He was educated in the public schools of Chicago and at the Rush Medical College, whence he was graduated in 1879, when not yet twenty-one years of age.

After graduation Dr. Moyer served for a short time as assistant physician at the Cook County Infirmary and then for about two years as assistant physician in the Illinois Eastern Hospital for the Insane at Kankakee, Ill. Following this service he studied abroad for about eighteen months, working principally at Heidelberg and Berlin and paying especial attention to nervous and mental diseases.

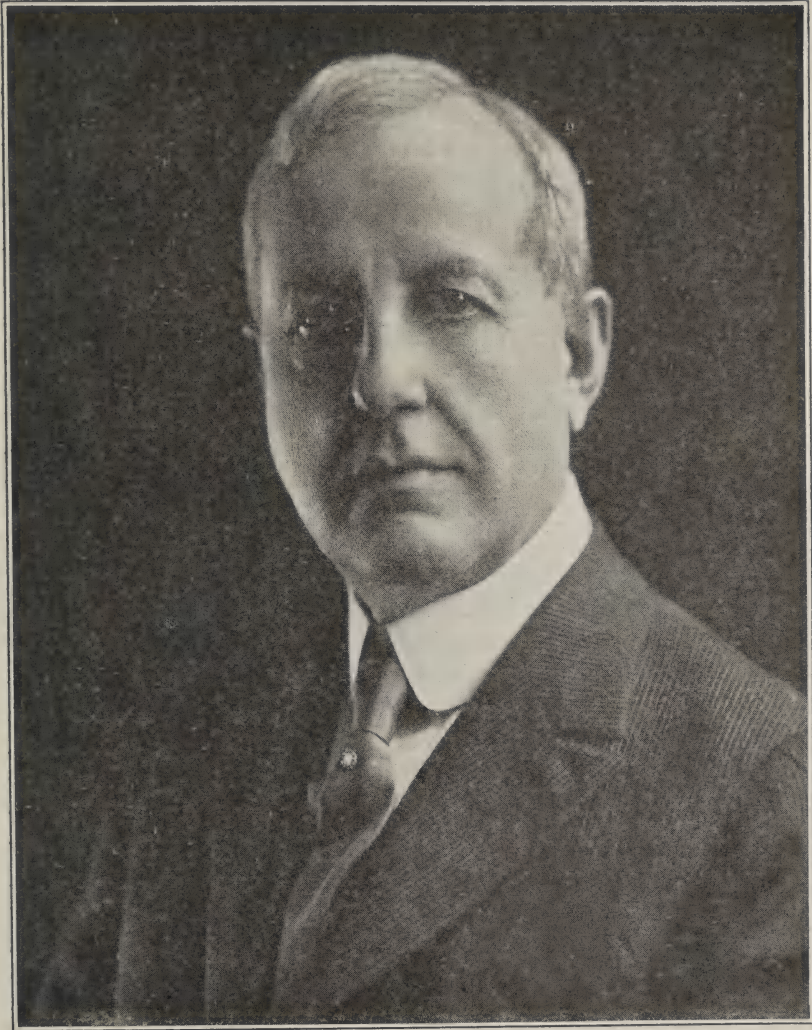
On his return to Chicago, Moyer immediately became very active in teaching and in practice. At Rush Medical College he was first lecturer on histology and then in succession lecturer on and professor of physiology, assistant to the chair of nervous diseases and adjunct professor of medicine. He was also neurologist to the Central Free dispensary (Rush Medical College) and later chief of the neurological clinic. He remained at Rush Medical College as a teacher for nineteen years, during much of which time he was on the staff of the Cook County Hospital, where he gave clinics, principally relating to nervous diseases but also on internal medicine. He served at different times as professor of nervous diseases at the Post Graduate School and as professor of nervous and mental diseases at the Chicago Clinical School. And for a time he was lecturer on railway medical jurisprudence in the Kent College of Law.

In 1888 he was appointed County Physician and as such had charge of the "Detention Hospital," now the Psychopathic Hospital. For most of the last forty years Dr. Moyer had been connected with this institution in one capacity or another and for several years preceding his death was chief of the visiting staff. He also served as attending neurologist at St. Luke's, the Columbus and Mercy Hospitals.

From its foundation in 1895 to the time of its consolidation with the *Therapeutic Gazette* in 1907, Dr. Moyer was the editor of *Medicine*, an excellent monthly journal.

From the time of his assistantship at Kankakee, Dr. Moyer was a liberal contributor to medical literature. He published about one

hundred and fifteen papers, most of which related to neurology, psychiatry or forensic medicine but many of them to internal medicine. To indicate the catholicity of his interests we might mention



DR. H. N. MOYER

A Case of Injury from Strong Electric Current (1886), Pilocarpin in Pulmonary Oedema (1889), Catarrhal Gastritis (1892), A Case of Colchicum Poisoning (1894), Skotography: Professor Roentgen's Discovery (1896), Notification and State Supervision of the Tuber-

culous (1900), The Relation of the Medical Editor to Original Communications (1901), Circumcision in Restricting the Spread of Syphilis (1901), Medical Graft (1903), and John Abercrombie, the First Neurologist (1917).

Soon after Dr. Moyer's return from Europe, circumstances introduced him to medico-legal work. He and others quickly discovered that temperament, taste and intellectual equipment adapted him to this pursuit. In less than ten years he probably was the best and perhaps the best known medical expert in Chicago. In spite of diversified interests and a good consultation practice this work grew with time so that for the later years of Dr. Moyer's life it constituted the major part of his practice. His knowledge of the law being extensive and accurate, his experience of courts and juries wide and deep, his judgment sound and his probity absolute, he was a rare adviser in matters of forensic medicine.

Probably because of his legal attainments and connections Dr. Moyer was at different times active and most effective in framing and passing various medical bills as well as in defeating such vicious measures as are constantly coming before our legislative bodies. He was also influential in legislation pertaining to the expert witness.

For many years Dr. Moyer was chairman of the medico-legal committee of the Illinois State Medical Society and for several years served the Chicago Medical Society in the same capacity. Certainly no one in the state was a better expert on medical liability and malpractice and Moyer's service to the profession never can be estimated. For years this service was cheerfully given with unmeasured generosity and remains a monument to his bigness of heart and mind.

Although Moyer was all his life an active worker, did many things well and leaves behind a record of high attainments, what he was overshadowed what he did. His qualities were those that endear one to his fellow men. He was of the tribe of Abou Ben Adhem. Though his intellect could not let him blink the bad, his eye ever seemed to be on the good. All manner of men were his friends because all manner of men had some appeal for him. He had the precious give of admiration and the broad charity that covers the blemishes of others.

Being fond of good food, good drink and good company, never taking himself too seriously, Moyer was the most companionable of men. He was entertaining and liked to be entertained. But he was more than a pleasing, adaptable companion. He was a loyal, never-failing friend, always an asset of his community, a high-minded gentleman.

HUGH T. PATRICK

G. DENY

The death of Dr. G. Deny denotes the loss of a figure active in the field of psychiatry and especially in the study of dementia precox. He has been known through his work upon the *Semaine médicale*, to which he was also a contributor of reports of the proceedings of scientific societies and of the Congress of Alienists and Neurologists. He was formerly physician to the Salpêtrière Hospital. He had also served under Péan in the St. Louis Hospital. With Péan he had published a work "*Le pincement des vaisseaux*," surgery being the branch of medicine to which he first gave his interest.

LEOPOLD LÖWENFELD

The death of Leopold Löwenfeld at the age of seventy-seven removed from the field of neurology a man widely known through his long years of service in this field. This service was made extensive through his activity in neurological literature. In his earlier years he was a collaborator upon the *Münchener med. Wochenschrift* and in later years he was a frequent contributor to its pages. His literary ability as well as his high ethical ideal was made evident in his work upon marital happiness, which first appeared anonymously. This is only one of a number of important works from his pen.


EUGEN VON MALAISÉ

A life of greatest practical usefulness has been cut off in its midst by the death after a short illness of Eugen von Malaisé in Munich, December, 1923. His fellow workers as well as the large number benefited by his wide activities join with his friends in lamenting his loss. He brought to his work a concentrated interest upon the practical clinical side, which he carried into every form of his activity. He secured earlier in life just the training which made it possible to carry out this ideal. Born in lower Bavaria in 1875 he received his medical education in Erlangen and Munich. After receiving his degree he worked with Bauer in the II. Medical University Clinic in Munich and then in Berlin under Oppenheim in the Neurological University Polyclinic. He enjoyed a particularly rich experience in clinical study in the hospitals of Paris where he studied under Pierre Marie and Babinski.

He settled in Munich as a neurologist and in 1910 was admitted to the medical faculty upon the presentation of his work upon the

disturbances of gait in the aged. His publications, with which should be mentioned the many produced by others under his inspiration and guidance, are many in number and of great importance in the entire field of organic neurology. He was occupied at the time of his illness and death with the relation of the physiology of the muscles to the endocrinous system. He had written much upon the peripheral nerves, a field where he had secured the collaboration of the orthopedist. He had been much occupied with the study of multiple sclerosis, syringomyelia, encephalitis, myasthenia, and with syphilogenous diseases of the central nervous system. Regarding the latter he was contributing a comprehensive study to Paul Mulzer's *Lehrbuch der syphilitischen Erkrankungen in der Allgemeinpraxis*. He had done particularly serviceable work in neurological surgery.

He rendered signal service to the wounded during the war. He succeeded, in 1923, in establishing a clinical neurological division in the leading hospital in Munich. He was aided in this by a number of influential men, among them the German-American philanthropist Heckscher. The service of this institution is to be extended first of all to victims of the war. It is to be a center also of neurological research as well as of aid to the suffering.



N. B.—All business communications should be made to Journal of Nervous and Mental Disease, 64 West 56th St., New York.

All editorial communications should be made to Dr. Smith Ely Jelliffe, Managing Editor, 64 West 56th St., New York.

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ORIGINAL ARTICLES

BLOOD ANALYSIS AND SUGAR TOLERANCE TESTS IN MENTAL DISEASE

By THOMAS M. BARRETT, M.D.

AND

PAUL SERRE

DIXMONT HOSPITAL, PENNSYLVANIA

In the following paper the results obtained from the blood examination of a number of patients suffering from mental disease are given in full, that is the figures are recorded from every separate case. Work of this kind has been brought more within the facilities of the ordinary laboratory through the method devised by Folin and Wu¹ in which a single blood filtrate may be used for making several determinations.

Using their method we have determined the amounts of non-protein nitrogen, urea, uric acid, creatinine and sugar in the blood of twenty cases of manic depressive psychosis and in twenty of dementia precox. Special care was taken to follow directions closely and every effort has been made to have our results as accurate and reliable as possible.

With one exception (L.M. No. 34, Table 1—Diabetes) the patients used were free from physical disease. There were two cases of mild hyperthyroidism (B.D. No. 1, and L.R. No. 10, Table 1) and twelve subjects were thin and underweight; most of these were in a state of depression with the usual tendency to undernourishment.

As is shown in Table No. 1, our results were fairly well within the normal limits. This has been the experience of others who have

¹ Folin, O., and Wu, H. A system of blood analysis. J. Biol. Chem., XXXVIII, 81, May, 1919.

made similar tests in mental cases. (Uyematsu and Soda,² Bowman,³ and Newcomer.⁴) Blood analyses for the forty cases gave us the figures recorded in Table 1.

The average of all the findings for both disease classes gives the following:

	Mg. per cent				
	Nonprotein nitrogen	Urea	Uric acid	Creati- nine	Sugar
Manic depressive psychosis.....	33.4	13.5	2.2	1.3	109
Dementia precox	33.8	16.2	2.3	1.4	114
Total average	33.6	14.9	2.3	1.4	112

Sugar Tolerance Tests: While engaged in the nitrogen and sugar determinations we found it convenient to perform the glucose tolerance tests in the same cases, repeating the test, after an interval of time, in sixteen instances, and, with a few additional cases, bringing the total number up to sixty; thirty of these were manic depressive psychosis and thirty dementia precox patients.

The following procedure, after the method of Janney and Isaacson,⁵ was carried out in each case: The patient was weighed and allowed no food after 7 P.M. At 7 A.M. the next morning blood was taken and the patient was then given a definite quantity of pure dextrose (1.75 gm. per kilo of body weight) in a measured quantity of water (2.5 c.c. per gm. of dextrose). Blood was taken at the beginning of each of the three following hours. We used 10 c.c. Luer syringes with fine needles and chose the same vein and point of entrance as often as possible. The patients were kept in bed in a room of even temperature. A sample of urine was collected, when it was possible, at the end of the first hour. The sugar determinations were made according to the last method described by Folin and Wu.⁶ For the readings we used a Duboscq pattern of colorimeter and a Klett lamp.

In Tables Nos. 2 and 3, which give the figures obtained in the manic depressive and dementia precox groups, respectively, we have made note of each patient's age and physical condition, the duration of mental illness, the type or phase of disease, the reaction to vein

² Uyematsu, S., and Soda, T. Blood analysis in cases of Catatonic Dementia Praecox. *J. NERV. & MENTAL DIS.*, 53, 367, May, 1921.

³ Bowman, K. Schizophrenic deterioration: Biochemical studies and presentation of case. *Am. Arch. Neurol. & Psychiat.*, 7, 395, March, 1922.

⁴ Newcomer, H. Blood constituents and mental state. *Am. J. Psychiat.*, 1, 269, April, 1922.

⁵ Janney, N., and Isaacson, V. A blood sugar tolerance test. *J. A. M. A.*, 70, 1131, April 20, 1918.

⁶ Folin, O., and Wu, H. A system of blood analysis. Supplement 1, *J. Biol. Chem.*, 41, 367, March, 1920.

TABLE NO. 1. BLOOD ANALYSIS

No.	Name	Age	Duration of mental disease	Physical condition	Mental disease	Milligrams per cent				
						Total non-protein nitrogen	Urea	Uric acid	Creatinine	Sugar
1	R. D.	22	18 Mo.	Hyperthyroidism	Man. Dep. Depressed	39	18	1.5	9	100
2	S. A.	69	7 Mo.	Underweight	Man. Dep. Depressed	33.3	15.8	1.2	1.2	105
3	A. G.	56	6 Yr.	Underweight	Man. Dep. Depressed	42.7	21.4	2.2	1.2	122
4	H. B.	48	1 Yr.	Underweight	Man. Dep. Depressed	33.3	15	3.3	1.1	99
5	E. S.	54	7 Yr.	Underweight	Man. Dep. Depressed	46.1	28.7	2.3	1.2	93
6	M. G.	54	2 Yr.	Underweight	Man. Dep. Depressed	37.9	17.5	2.5	1.7	100
7	J. D.	25	3 Wk.	Good	Man. Dep. Depressed	38	18.9	1.6	1.6	121
8	M. L.	46	3 Mo.	Underweight	Man. Dep. Depressed	44	15	2.5	1.7	132
9	L. D.	30	9 Mo.	Underweight	Man. Dep. Depressed	26	12	1.5	1.1	82
10	L. R.	36	9 Mo.	Hyperthyroidism	Man. Dep. Depressed	42	15.7	2.1	1.1	90
11	R. E.	50	1 Yr.	Good	Man. Dep. Depressed	33.3	16.4	1.7	1.2	94
12	R. M.	25	7 Wk.	Good	Man. Dep. Depressed	50	22	2.7	1.3	105
13	H. J.	70	6 Wk.	Good	Man. Dep. Depressed	32	16.4	1.2	1.5	107
14	C. S.	42	2 Yr.	Good	Man. Dep. Depressed	35	21.4	2.5	1.1	107
15	F. F.	42	7 Mo.	Good	Man. Dep. Depressed	30	14	1.7	2	125
16	M. W.	56	10 Mo.	Good	Man. Dep. Manic	31.9	15	3.3	1.3	111
17	A. Z.	33	3 Mo.	Good	Man. Dep. Manic	37.5	14.3	2.1	1.2	105
18	E. S.	48	12 Yr.	Good	Man. Dep. Manic	37	16.4	2.1	1.1	125
19	A. B.	37	2 Yr.	Good	Man. Dep. Manic	55	30	2.3	1.3	123
20	S. H.	23	3 Mo.	Good	Man. Dep. Manic	36.8	17	2.5	1.6	131
Average						33.4	13.5	2.2	1.3	109
21	E. H.	25	5 Yr.	Good	Dem. Precoc. Hebe.	30	13	1.6	9	105
22	R. N.	26	18 Mo.	Good	Dem. Precoc. Hebe.	32	15	1.5	1.2	100
23	H. H.	30	10 Yr.	Good	Dem. Precoc. Hebe.	30	12.5	2.1	1.1	116
24	H. C.	27	3 Yr.	Good	Dem. Precoc. Hebe.	36	16.6	2.1	1.1	109
25	A. C.	32	3 Yr.	Good	Dem. Precoc. Hebe.	33	13.8	2.5	1.3	103
26	M. B.	38	8 Yr.	Good	Dem. Precoc. Hebe.	34	18	2.5	1.3	142
27	A. P.	35	5 Yr.	Underweight	Dem. Precoc. Hebe.	44.5	25	2.5	1.1	105
28	J. P.	43	12 Yr.	Underweight	Dem. Precoc. Hebe.	34	12	3.1	1.7	127
29	F. P.	26	5 Yr.	Good	Dem. Precoc. Hebe.	30	16.6	2.2	1.5	138
30	E. S.	31	5 Yr.	Good	Dem. Precoc. Para.	37.5	16	2	1.2	101
31	O. C.	34	7 Yr.	Good	Dem. Precoc. Para.	32	16.6	1.6	1.5	100
32	M. F.	34	5 Yr.	Good	Dem. Precoc. Para.	32	16.2	2.5	2	100
33	G. A.	29	4 Yr.	Good	Dem. Precoc. Para.	37	16	2.3	2.2	103
34	L. McC.	58	5 Yr.	Good	Dem. Precoc. Para.	38	18	4.1	1.2	204
35	M. S.	19	1 Yr.	Fairly good	Dem. Precoc. Para.	30	16	2.5	1.2	124
36	E. G.	31	18 Mo.	Underweight	Dem. Precoc. Cata.	40	17.5	2.6	1.4	95
37	N. S.	20	18 Mo.	Underweight	Dem. Precoc. Cata.	45	25	2.9	1.5	83
38	E. P.	28	1 Yr.	Underweight	Dem. Precoc. Cata.	24	11	2	1.4	98
39	S. M.	38	1 Yr.	Fairly good	Dem. Precoc. Cata.	26	14	1.9	1.4	105
40	J. P.	37	4 Yr.	Good	Dem. Precoc. Cata.	52	15	2.6	1.4	122
Average						33.8	16.2	2.3	1.4	114

puncture, and the amount of sugar in milligrams for the four consecutive hours. The subjects were all women. Under "physical condition" we have noted the state of the patient's nutrition. The "emotional reaction to vein puncture" was recorded because of the possible influence of the emotional state on the sugar curve.

Manic Depressive Group—Table No. 2: The group included twenty-four patients in the depressed phase of the disease and six in the manic phase.

Those in the depressed phase are placed first, not only because they constitute the larger number of cases, but also because in them deviation from normal tolerance is more pronounced. Our results differentiate them into two groups; from one in which anxiety and mental distress was quite marked, we obtained a high blood sugar content at the end of the first hour (in most instances reaching 200 and over); in the other group where the depression was comparatively mild, the sugar curve is fairly within the normal limits.

Of the six manic cases it was found that only one showed a decided hyperglycemia after ingestion of dextrose. This was a recent case with considerable emotional excitement (E.S. No. 30, Table No. 2).

The averages for the components of the manic depressive group are given in the following table, division being made on the basis of emotional reaction:

MANIC DEPRESSIVE PSYCHOSIS				
	Fasting	1st hour	2nd hour	3rd hour
Depressed phase—anxious type.....	110	198	162	113
Depressed phase—mild dep.	100	123	104	101
Manic phase—hypomania	123	116	109	91
Total averages	111	145	125	102

These results are in harmony with those of Kooy,⁷ who observed that in marked emotional depression the sugar curve reached an unusually high point near the end of the first hour, while in the mild types of mania and depression the elevation was not nearly so pronounced.

But these averages, in the present state of our knowledge, have little more than an arithmetical value. Study of the tables shows us that the sugar tolerance follows individual variations about which averages tell us nothing. Consider numbers 2 and 6 in the anxious depressed group, cases which have identical histories in all particulars except age and amount of underweight. They have, however,

⁷ Kooy, F. Hyperglycemia in Mental Disease. *Brain*, 42, 214, 1919.

TABLE NO. 2. SUGAR TOLERANCE TEST
MANIC DEPRESSIVE GROUP

No.	Name	Date	Weight lb.	Age	Duration of mental disease	Physical condition	Type of mental disease	Emotional reaction during vein puncture	Milligrams per 100 c.c.				Urine
									Fasting	1st hour	2nd hour	3rd hour	
1	B. D.	10-1-21	102	22	18 Mo.	Hyperthyroidism	Anxious Dep.	Slight	100	153	136	113	Negative
2	S. A.	10-18-21	88	69	7 Mo.	Underweight	Anxious Dep.	Marked	105	200	163	90	"
3	A. G.	10-25-21	79	56	6 Yr.	Underweight	Anxious Dep.	Marked	122	235	125	100	"
4	Same	3-2-22	78.5	56	6 Yr.	Underweight	Anxious Dep.	Marked	118	175	173	129	"
5	H. B.	11-15-21	85	48	1 Yr.	Underweight	Anxious Dep.	Marked	99	148	119	91	"
6	E. S.	12-20-21	65	54	7 Mo.	Underweight	Anxious Dep.	Marked	93	250	250	105	"
7	M. G.	12-27-21	76	54	2 Yr.	Underweight	Anxious Dep.	Marked	100	208	172	60	"
8	J. D.	1-31-22	104.5	25	3 Wk.	Good	Anxious Dep.	Marked	121	222	215	194	"
9	M. L.	2-2-22	78	46	11 Mo.	Underweight	Anxious Dep.	Marked	132	238	206	80	"
10	Same	8-22-22	79	46	5 Mo.	Underweight	Anxious Dep.	Slight	111	167	105	85	"
11	M. L.	6-27-22	118	35	2 Yr.	Good	Anxious Dep.	Slight	143	222	118	118	"
12	H. J.	8-8-22	93.5	70	6 Wk.	Good	Anxious Dep.	None	107	201	156	125	"
13	C. S.	8-8-22	122	42	2 Yr.	Good	Anxious Dep.	Slight	107	166	162	148	"
14	L. J.	8-19-22	90	51	4 Mo.	Underweight	Anxious Dep.	Marked	94	167	111	105	No sample
15	A. McC.	8-29-22	108½	52	5 Yr.	Good	Anxious Dep.	Slight	104	222	215	147	Negative
								Average	110	198	162	113	
16	L. D.	11-22-21	106	30	9 Mo.	Underweight	Mild Dep.	None	82	71	80	60	No sample
17	L. R.	12-3-21	137.5	36	9 Mo.	Hyperthyroidism	Mild Dep.	Slight	90	133	100	90	Negative
18	S. E.	12-6-21	138	50	1 Yr.	Good	Mild Dep.	None	94	84	133	95	"
19	Same	2-14-22	137	50	1 Yr.	Good	Mild Dep.	None	98	148	105	143	"
20	Same	5-2-22	145	50	1 Yr.	Good	Mild Dep.	None	100	169	105	132	"
21	R. M.	1-10-22	107	25	1 Wk.	Good	Mild Dep.	Slight	105	116	111	125	"
22	Same	2-28-22	108	25	7 Wk.	Good	Mild Dep.	Slight	118	125	80	74	"
23	F. F.	6-27-22	123	42	7 Mo.	Good	Mild Dep.	Slight	125	111	109	100	"
24	S. C.	8-26-22	134	32	4 Mo.	Good	Mild Dep.	Slight	87	152	111	88	"
								Average	100	123	104	101	
25	M. W.	10-6-21	163.5	56	10 Mo.	Good	Hypomania	None	111	82	106	90	Negative
26	A. Z.	10-11-21	111½	33	3 Mo.	Good	Hypomania	None	105	114	100	72	"
27	A. B.	2-22-22	97	37	2 Yr.	Good	Hypomania	Slight	123	69	96	112	"
28	Same	5-16-22	98.5	37	2 Yr.	Good	Hypomania	Slight	144	91	67	103	Alb. (trace)
29	S. H.	6-6-22	110	23	3 Yr.	Good	Hypomania	Slight	131	133	137	83	Negative
30	E. S.	2-21-22	126	48	6 Wk.	Good	Acute Mania	Slight	125	210	148	88	
								Average	123	116	109	91	
								Total average	111	145	125	102	

extremely different sugar curves. In spite of an age difference of fifteen years in favor of number 6 she has the far more abnormal curve. However, the advantage in age is, at this time of life, perhaps not so important, and the disadvantage suffered by number 2 in respect to age may well be more than compensated by her slighter degree of undernourishment.

The extremely low weight of sixty-five pounds in number 6 points, in the circumstances, to a greatly diminished tissue metabolism and we might well expect to find, as we do find, a long sustained excess of sugar in the blood.

Number 2 and number 7 have approximately identical curves; number 2 is sixty-nine years old and has been seven months ill; number 7 is fifty-four years old and has been two years ill; in other respects their legends are the same. In these cases we might conjecture that the greater age of the one patient was equalized by the shorter time of illness of the other, making them react identically to excess sugar feeding. But the true explanation is probably quite a different one.

Even supposing that in these patients intolerance to excess sugar feeding is related to their psychosis through a common origin in some glandular defect, it is in accord with all probabilities to believe that a good proportion of them may have other incidental or associated gland defects which act either as aggravating or compensatory forces in controlling sugar tolerance. If this were so it would furnish an explanation of identical curves obtained from patients with very different histories and *vice versa*. And, since functional gland disturbances come and go rapidly, we might, through such disturbances, get extremely different sugar curves at different times from a patient whose mental and physical condition had not visibly changed.

Apart from the different types of their disease, and their differing irritability under slight stimulus, our manic depressive group includes age variations from twenty-two to seventy years, weight variations from 65 to 164 pounds, and variations in length of sickness from three weeks to six years. So great a lack of uniformity in conditions—a lack probably much greater in conditions that we are unable to measure than in those which our case histories record—makes “averages” of sugar tolerance often represent little more than mathematical fictions. What, for example, do we learn from the mean values of two different curves obtained from the same patient? Our mean represents nothing actual. For the present two such curves must be accepted as facts of which the interpretation is lacking, and their “average” represents no concrete value.

TABLE NO. 3. SUGAR TOLERANCE TEST
DEMENTIA PRECOX GROUP

No.	Name	Date	Weight	Age	Duration of mental disease	Physical condition	Type of mental disease	Emotional reaction during vein puncture	Milligrams per 100 c.c.				Urine
									Fast-ing	1st hour	2nd hour	3rd hour	
31	E. H.	11-22-21	118 lb.	25	5 Yr.	Good	Hebephrenic	Slight	100	125	74	74	Negative
32	Same	5-9-22	118	25	5 Yr.	Good	Hebephrenic	Slight	108	116	110	103	Alb. (trace)
33	R. N.	12-2-21	119	26	18 Mo.	Good	Hebephrenic	None	100	111	92	111	Negative
34	Same	4-4-22	137	26	22 Mo.	Good	Hebephrenic	None	107	190	200	107	"
35	E. H.	12-20-21	138	30	10 Yr.	Good	Hebephrenic	None	116	118	120	116	"
36	Same	4-4-22	127	30	10 Yr.	Good	Hebephrenic	None	125	169	138	125	"
37	H. M.	1-17-22	118	27	3 Yr.	Good	Hebephrenic	Marked	109	182	133	131	"
38	A. C.	1-17-22	126	32	3 Yr.	Good	Hebephrenic	None	103	103	108	88	"
39	Same	5-9-22	125	32	3 Yr.	Good	Hebephrenic	None	104	130	113	99	"
40	M. B.	4-18-22	95 5	32	8 Yr.	Good	Hebephrenic	None	142	235	170	107	No sample
41	Same	5-15-22	100	32	8 Yr.	Good	Hebephrenic	None	114	148	133	114	Alb. (trace)
42	A. P.	4-25-22	90	38	5 Yr.	Underweight	Hebephrenic	None	105	135	133	102	Negative
43	J. McC.	4-25-22	175	43	12 Yr.	Good	Hebephrenic	None	127	117	128	80	"
44	F. F.	5-3-22	176	26	5 Yr.	Good	Hebephrenic	None	138	190	160	133	"
								Average	114	148	129	106	
45	E. S.	12-13-21	117	31	5 Yr.	Good	Paranoid	None	101	100	67	85	Negative
46	Same	2-28-22	112	31	5 Yr.	Good	Paranoid	None	92	118	140	100	"
47	O. C.	1-24-22	113	34	7 Yr.	Good	Paranoid	None	100	161	132	93	"
48	Same	4-11-22	113	34	7 Yr.	Good	Paranoid	None	143	143	149	128	"
49	M. F.	1-24-22	106	34	5 Yr.	Good	Paranoid	None	100	135	125	111	"
50	Same	4-11-22	105.5	34	4 Yr.	Good	Paranoid	None	99	138	138	103	"
51	G. A.	2-7-22	163	29	4 Yr.	Good	Paranoid	Marked	103	122	148	103	Sugar (trace)
52	Same	6-6-22	164	29	4 Yr.	Good	Paranoid	None	105	222	185	149	Ind. (trace)
53	D. C.	8-26-22	99	21	8 Yr.	Good	Paranoid	None	100	145	139	95	Negative
								Average	105	143	126	104	
54	M. S.	10-4-21	119	19	1 Yr.	Underweight	Catatonic	Marked	124	152	129	95	Sugar (trace)
55	E. G.	12-6-21	87.5	31	13 Mo.	Underweight	Catatonic	Marked	95	200	103	65	No sample
56	A. P.	1-3-22	85	28	1 Yr.	Underweight	Catatonic	Marked	98	201	153	100	"
57	S. M.	1-3-22	96	38	1 Yr.	Underweight	Catatonic	Slight	105	100	129	105	Negative
58	J. P.	4-18-22	107	32	4 Yr.	Good	Catatonic	None	122	173	145	121	"
59	Same	5-23-22	110	32	4 Yr.	Good	Catatonic	None	98	87	125	74	"
60	N. S.	8-22-22	100	21	2 Yr.	Good	Catatonic	None	121	138	133	124	No sample
								Average	109	150	131	96	
								Total average	109	147	129	102	

Dementia Precox Group—Table No. 3: In this group of thirty patients the diagnosis of dementia precox was fairly certain, but some difficulty was experienced in making a satisfactory division as to the type of the disease. The catatonic types were not hard to distinguish but some doubt must be admitted as to the hebephrenic and paranoid types where the points of distinction were somewhat obscured because of dementia and long duration of the disease.

Unlike the findings of other recent work⁸ our results give a great variety of sugar curves but none that may be termed typical nor of constant occurrence. We found, by repeating the test after an interval of time, that although the patient had apparently remained in the same condition, the curve in most cases differed greatly from the first one. We also found that a number of patients in whom there appeared to be decided apathy and lack of emotion gave, at the end of the first hour, a blood sugar curve of considerable height.

The averages of the dementia precox group are as follows:

DEMENTIA PRECOX				
	Fasting	1st hour	Mg. per cent	
			2nd hour	3rd hour
Hebephrenic type	114	148	129	106
Paranoid type	105	143	126	104
Catatonic type	109	150	131	96
Total averages	109	147	131	102

The average of intolerance toward an abnormally large glucose meal shown by our manic depressive cases may be compared with the average intolerance shown by our dementia precox cases in the following figures:

	Fasting	1st hour	2nd hour	3rd hour
Manic depressive psychosis.....	111	145	125	102
Dementia precox	109	147	131	102

For the dementia precox group, in spite of the fact that most of the curves are fairly normal, it seems scarcely less fallacious to speak of averages than for the manic depressive group. Take the case of number 33 who gave, eighteen months after the onset of illness, a fairly normal curve, and four months later a very steep, sustained curve. All the conditions appear to have remained the same except that the patient has put on 18 pounds in that time. Since she was not underweight at the time of the first test this circumstance is perhaps indicative of lowered oxidation: the increased intolerance is to be expected, and we might, with some plausibility, suppose that the

⁸ Raphael, T., and Parsons, J. Blood Sugar Studies in Dementia Precox and Manic Depressive Insanity. *Am. Arch. Neurol. & Psychiat.*, 5, 687, June, 1921.

mean of her two curves would be that one which we should have got at a time nearly half-way between the times of obtaining the two actual ones; but the two curves given by E.H. (Nos. 35 and 36) show processes that invalidate our conjecture for the patient, otherwise unchanged, has lost some weight and shows less tolerance to excess glucose at the time of the second test. And had the two curves from number 33 been obtained from different patients, we should, in most cases, not be in a position to furnish even a tentative interpretation of their differences.

The two curves obtained from number 40 illustrate still more conspicuously a behavior which is the reverse of what we saw in number 33. The subject, thirty-two years old and eight years ill, gives first a curve showing an extreme sugar intolerance, followed a month later by a nearly normal curve. In the meanwhile, except for a slight gain in weight, no change has taken place in the patient's condition. Such rapid fluctuations in the sugar tolerance can naturally not be due to the constantly operating causes of dementia precox.

If we endeavor to relate sugar tolerance to degeneration of the cells of Leydig, and assume that increased intolerance for excess sugar will keep pace with the degradation of interstitial tissue we ought to expect that the early cases would furnish relatively normal curves and that the long standing cases would give steep, long sustained curves. Examination of the table brings out that one of the most abnormal of the thirty curves (No. 34, second test of R.N.) comes from a patient of twenty-six years, ill for twenty-two months, whereas number 43, who is forty-three years old and ill for twelve years, has a normal curve.

In a paper published since the completion of our work, Bowman⁹ gives the results of sugar tolerance tests in ten cases of dementia precox. He found normal curves in three cases, sustained curves in three cases, and, in two cases, curves which were sustained at the first test and normal at the second test-made six months later. Bowman says: "As a result of biochemical studies on ten cases of dementia precox no constant findings were obtained which would serve to explain dementia precox on the basis of a simple dysfunction of a single gland. There was found a tendency towards a low basal metabolism and an abnormally sustained blood sugar curve but such

⁹ Bowman, Carl M., Eidson, Joseph P., and Burladge, Stanley P. *Biochemical Studies in Ten Cases of Dementia Praecox*. Bost. Med. and Surg. Jour., 187-358, Sept. 7, 1922.

findings were not constant. In no case was there an increased basal metabolism nor was there a subnormal (flat) sugar curve."

Even were the results less irregular, the number of findings so far published is manifestly too small to justify generalizations concerning the relation of dementia precox to sugar tolerance, or to establish a hope that the sugar tolerance test may at present be usefully employed as a diagnostic procedure. Our findings and Bowman's go to show that no constant relation between dementia precox and sugar tolerance has yet been observed. If a relation exists it is seemingly influenced by factors which create fluctuations in sugar metabolism that allow large quantities to be disposed of at times and at other times depress the processes of sugar disposal. An explanation of these variations, if they are intrinsically related to the dementia precox state, must presumably wait upon a study of the causes responsible for the disease.

In the meantime, since no great number of sugar tolerance tests have yet been made, and particularly since the results so far published reveal irregularities, inconsistencies and disagreements, it has seemed appropriate to publish the results of our analysis *in extenso*. Only when this is done can we compare, even approximately, the results obtained by different investigators and seek intelligently for the origin and meaning of their variable, often opposed, findings.

So many and such complex factors are involved that all available data are needed before we can hope to evaluate the seemingly opposed evidence concerning sugar tolerance in mental disease.

THE OCULOCARDIAC REFLEX AND THE ARTERIAL TENSION IN HYDROCEPHALUS

BY DR. J. ROUBINOVITCH

PHYSICIAN AT THE HOSPITAL AT BICÊTRE

AND

DR. O. TIDEMAND-JOHANNESSEN

SENIOR INTERN OF THE HOSPITALS AT CHRISTIANIA, NORWAY

We have sought to gain an idea of the oculocardiac reflex in hydrocephalus in its relations to arterial tension. To this end we examined 12 hydrocephalic patients in the children's service at the hospital at Bicêtre with the aid of the manometric oculocompressor invented by one of us and by means of the tensiphone Laubry-Vaquez. The results are as follows:

First of all one may divide the twelve cases into two groups:

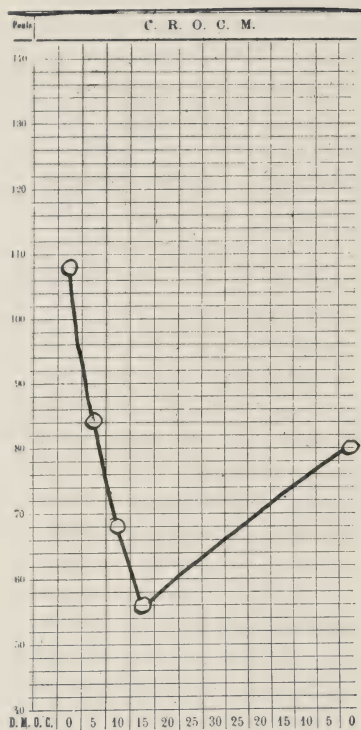
1. A first comprising 8 similar cases.
2. A second comprising 4 cases.

In the chief group two interesting details may be noted, first a very exaggerated brachycardia and then persistence of the brachycardia even after the compression has ceased.

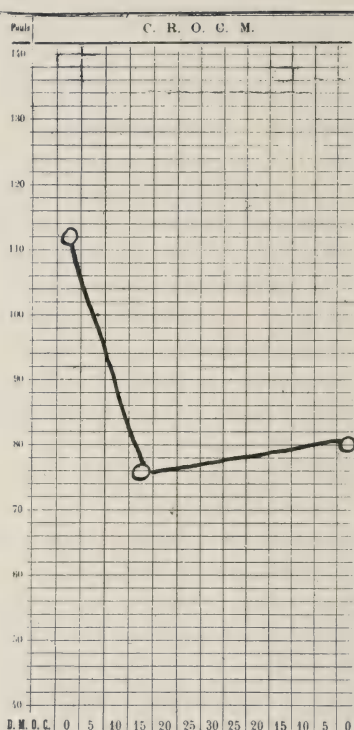
- Examples of No. 1. Pulse 108 with no compression.
 Pulse 56 with an amount of compression
 to 15 cm. of Hg.
 Pulse 80 two minutes after compression.
- No. 2. Pulse 112 to zero.
 Pulse 76 to 15 cm. of Hg.
 Pulse 80 to zero two minutes after.
- No. 3. Pulse 96 to zero.
 Pulse 70 to 15 cm. of Hg.
 Pulse 78 to zero two minutes after.
- No. 4. Pulse 96 to zero.
 Pulse 66 to 15 cm. of Hg.
 Pulse 76 to zero two minutes after.

The four other cases gave similar results.

The first group:



Nom: J. No. 1. Diag. Hydroceph.
Dates: 15-11-20. 11 years



Nom: No. 2. Diag. Hydroceph.
Dates: 11-11-20. 10 years

The second group presents very different results. In the four cases at the beginning with a weak compression there was noted at first an acceleration of the pulse, then a bradycardia when the compression became stronger, finally a marked acceleration when the compression of the eyes had been stopped.

Examples: No. 1. Pulse 84 to zero.
Pulse 92 to 10 cm. of Hg.
Pulse 64 to 15 cm. of Hg.
Pulse 92 to zero two minutes after.

No. 2. Pulse 72 to zero.
Pulse 80 to 10 cm. of Hg.
Pulse 72 to 15 cm. of Hg.
Pulse 92 to zero two minutes after

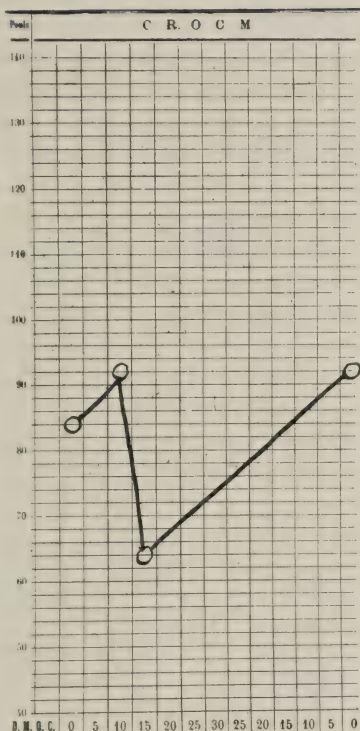
No. 3. Pulse 60 to zero.

Pulse 64 to 10 cm. of Hg.

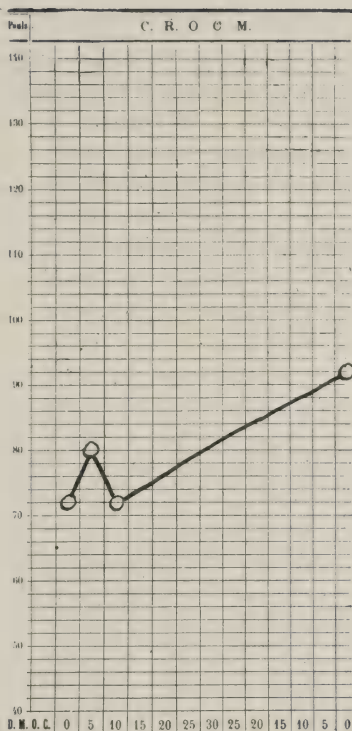
Pulse 52 to 15 cm. of Hg.

Pulse 76 to zero two minutes after.

The second group:



Nom: A. C. Diag. Hydroceph.
Dates: 4-11-20. 10 years



Nom: T. B. Diag. Hydroceph.
Dates: 6-11-20. 9½ years

But the most interesting feature of these researches seemed to be the diminution of the arterial tension taken at the level of the bend of the elbow during the compression of the eyes. This fact is more often found among the cases of gross hydrocephalus than in the cases of slight hydrocephalus. Yet we have met it in almost all the cases which we have studied.

Already with a compression of 10 cm. of Hg. the pulse begins to grow weaker, at 15 cm. of Hg. it is thready and very difficult to count and at the same time the child begins to complain saying that the compression makes it feel badly. Thus one child presented very

clear lipothymic symptoms with a compression of 15 cm. of Hg. An analogous fact, syncope with stoppage of the heart 4 seconds, has already been noted June, 1914, by MM. Roubinovitch and de la Soudière in a case of hydrocephalus.¹ The arterial pressure which we have measured with the tensiphone Laubry-Vaquez has always given results of which the maximum has never been greater than $14\frac{1}{2}$ or the minimum less than 6. In general the maximum has varied from $14\frac{1}{2}$ to 10 and the minimum from 12 to 6. With two cases of very large hydrocephalus it was very difficult to perceive the pulsations.

Two children presented the following pressures: maximum, 10; minimum, $7\frac{1}{2}$; one 14-12; one 14- $9\frac{1}{2}$; one $14\frac{1}{2}$ -9; two $13\frac{1}{2}$ -10; one 12-9; one $11\frac{1}{2}$ -8; one $10\frac{1}{2}$ -7; one 10-8; one 10-6.

In conclusion one may say that the oculocardiac reflex seems to show a rather special curve with hydrocephalics. But it appears necessary to examine many more children afflicted with this disorder in order to know if the curve is truly characteristic. In any case the important diminution of the strength of the arterial pressure under the influence of the ocular compression is interesting and deserves to have attention fixed upon it since it is much more accentuated in the different cases of hydrocephalus than with other arrested children. One may ask why one finds this diminution of arterial tension under the influence of ocular compression, as well as this extremely accentuated bradycardia among cases of gross hydrocephalus.

It must be noted that with hydrocephalics the pulsations of the radial artery are small, weak and difficult to count even without compression of the eyes. It is likely that the vascular system remains in a state of frustrated development in these patients. A sort of microsphygmus has also been spoken of and it is a well known fact that the venous circulation is little accentuated in the first period of hydrocephalus when almost all of the veins of the face are visible. With hydrocephalics the intracranial pressure is higher than with normal subjects and in consequence an ocular compression produces in them a new increase of pressure. If now one studies the anatomic disposition under the jugular foramen one finds that the pneumogastric is situated in front of the internal jugular vein and that below this that nerve descends in the groove which this vein and the internal carotid artery form for it.

The ocular compression increases the intracranial pressure with the result that the blood in the cranium circulates with more difficulty.

¹ Roubinovitch and de la Soudière: La Reflexe oculo-cardiaque dans les démences organiques. Société de Psychiâtrie, June 18, 1914.

This has as a consequence an increase of the volume of the internal carotid artery, as well as of the primitive carotid artery. The pneumogastric is thus compressed and irritated, the nerve which controls the slowing of the heart (pulse). If the pulse (heart) becomes much slower the arterial tension diminishes equally. In effect if the bradycardia is very marked the arterial tension is lowered for the volume of blood sent out by the heart is less. The volume of blood transported is equal to the product of the volume of each pulse beat multiplied by the number of the contractions per minute.²

Another hypothesis for explaining these phenomena may be set forth. One may admit that the ocular compression directly irritates the vagus by increasing the intracranial pressure and in particular the motor center of the pneumogastric nerve (nucleus ambiguus) and that this irritation produces a bradycardia.

In brief, it appears from the researches that we have undertaken that with hydrocephalics the ocular compression involves a bradycardia more intense and more persistent than with normal subjects as well as a diminution of the arterial tension.

² Cf. *Lehrbuch der Farmakologie*. E. Poulsson, Professor at the University, Christiania, p. 154, ed. 1914.

THE EVOLUTION OF VISION

BY ARISTOPH SPARE, M.D.

CHICAGO

The phenomena of the universe are either (1) coexistences—those manifested simultaneously or perceived in *any* order; and (2) sequences—those occurring successively or perceived in a *certain* order.

In the first instance, the relations of the phenomena toward one another are those of *Space*; in the second instance—those of *Time*. A *fixed* panoramic scene, for example, where objects are discretely distributed, presents an instance of coexistent phenomena only, and the implied relations are exclusively those of space. On the other hand, a series of sonorous vibrations, recurring at intervals, illustrates a case of mere sequential phenomena, and the involved relations are purely those of time.¹

The response of the conscious organism to phenomena and their relations is through the senses and the brain. If the disturbance is *peripherally* initiated it is called a sensation or *perception*; if *centrally* initiated—an idea or *conception*. As regards the origin of the *higher* conceptions of Space and Time, the keenest minds of intellectual Europe are radically divided over it. The issue is virtually one of Deduction versus Induction. On one side stand the transcendentalists, principally Germans, with Emmanuel Kant as leader, who maintain that the conceptions of Space and Time are *prior* to the sensations (*a priori*) and *determine* them; while on the other side are the empiricists, mostly English, with John Stuart Mill as exponent, who insist that the conceptions are *posterior* to the sensations (*aposteriori*) and are determined *by* them.

Discarding the further discussion of these elements as belonging to the higher branches of psychology, it may be stated in general terms, that the eye is the primitively specialized sense organ for the perception of space relations; while the ear is the primitively special-

¹ Coexistences and sequences form the "warp" and "woof" of the cosmic texture. The former express the *substantive* forces of nature; the latter—the *creative*. Considered in the abstract, coexistences are *passive aggregates* in a state of *isolation*; while sequences are *active changes* in a state of *causation*. The first are subject to the law of *gravitation*; the second—to the law of *evolution*.

ized sense organ for the appreciation of time relations. Hence it coexistences having *optic* attributes; while audition is a sensation which responds to sequences having *acoustic* properties. The *nascent* ideas therefore of "where" and "when" are studies of ophthalmology and otology, respectively.

How and under what circumstances this initial-rôle of the ocular apparatus becomes manifest, will be best understood by a study of its genesis and incipient development.

Considerations of the ear, not being relevant to this discussion, are henceforth discarded. An analogous mode of reasoning, however, can, with consistency, be applied also there.)

The evolution of any organ portrays in miniature those traits characterizing the evolutionary process in general. Any aggregate, whether planet, organic being, or specific organ, passes in the course of its development from a state of simplicity to a state of complexity; from a state of uniformity to a state of multiformity and from a state of indefinitely related parts to a state of definitely related parts.

The human eye, accordingly, represents a perceptive equipment which has developed from the original, uniform peripheral sensibility in the course of eons of time—a period coextensive with the biologic history of our planet. The *modus operandi* of the process consists of the acquisition, retention and ever-increasing accumulation of minute modifications.

Common peripheral body sensibility to contact of matter or vibrations is therefore primary, original and generic; while all other senses are secondary, derived and specific.

The derivative character of the senses was even suspected by the ancient Greeks. Democritus, the "laughing philosopher" (about 400 B.C.) expressed his belief that all the special senses have originated from the cutaneous tactile sense. But what was a mere belief with him is an established theory with us. All the special senses are, embryologically, developed from the ectodermic layer; and embryology, as was pointed out by Haeckel, reproduces, in a condensed, abbreviated form, all the changes and traits acquired by the species in the course of its biologic evolution. The pouching, therefore, of the anterior brain vesicle to form the primary optic vesicle; and the invagination of the latter to produce the secondary optic vesicle, are merely detailed embryologic facts which have identical implications.

Before differentiation sets in and while the structures are uniform or homogeneous, the light sensitiveness manifested is evenly diffused over the entire surface. All exposed parts are equally

affected. Whether this generalized reaction on the part of the organism is caused by heat or light waves—by thermic or luminiferous vibrations, is doubtful. The discrimination of forms under such conditions, even vaguely, is impossible. The sense of perception is limited only to an extremely feeble appreciation of general illumination and general obscurity. Consequently, the passage of an object can only be felt negatively: by the absence of those peripheral disturbances which light waves produce.

The first indications foreshadowing *form* perception are seen in the Planaria (Spencer). In this low biologic order, pigment granules appear at a certain spot of the common surface. The area thus endowed becomes, in consequence of its optical isolation, more sensitized and the response to light more ready and more intense.

The important fact to be remembered in connection with this feature is, that with *restriction* of the sensitive area, there goes on a corresponding *concentration* of function. Here, as elsewhere, the reciprocal relation between extension and intension—that the more extensive the sensitiveness the less intensive it is; and the more intensive the less extensive—is clearly observable. So that instead of being able to differentiate only in a feeble manner between general illumination and general obscurity, an organism having a restricted pigmented area, like the planaria for instance, can, in addition, appreciate the presence of any body which casts a shadow on the sensitive spot. However, the contrast between light and darkness, or between widely different shades of light, is about all such rudimentary vision can possibly recognize. But since distinct obscuration by small objects is only possible when they are very near, the character of the shadow serves as a sort of guide in the determination of the proximity or remoteness of the object; the nearer the object the more distinct the shadow cast; the further, the less distinct. And thus, Spencer concludes, that “the nascent idea of coexistence or space relation is synchronous with the evolution of sight” (Principles of Psychology).

A slight convexity of the dermic layer over the pigmented spot by a process of pouching or by a localized cell proliferation, making ray convergence possible, is a further step in the evolution of vision. Finer variations in quantity of light are appreciated, and the same object can be seen at greater distances.

A more symmetrical arrangement of the overlying tissues and a more homogeneous composition of its units is an additional advance along the same lines. For these factors are the early forerunners in the eventual production of pure, transparent refractive media.

The interpretation of all such changes from a biologic point of view is that they represent not only *structures* for *present purposes*, but likewise *foundations* for *future alterations*; that the various tissue metamorphoses manifested in the ascent of types, besides serving *proximately* as *functioning organs*, serve also *remotely* as *concentration points*, whence nature's forces take new departures for further advances along progressive lines.

We are still, however, at the very threshold of biologic creation. How are we to bridge the abysmal gap separating diffuse peripheral perception on one side, and the telescopic eye of the eagle or the stereoscopic vision of a Raphael on the other side? How is the evolutionary process to be traced from its beginning somewhere in the Laurentian formation—the earliest representative of the sedimentary deposits—through the entire series of geologic strata, to the present period? In short, how are we to reproduce, theoretically, the “visual-outfits” of the intermediate forms which nature was steadily evolving for millions of years?

To examine in detail the successive and divergent types through such a tremendous lapse of time, while undoubtedly associated with much interest and suggestion, is obviously out of place in a treatise of limited scope. The utmost that will be attempted in the following discussion will be to point out in a general way some of the main evolutionary features linking the different orders of the animal kingdom.

One fact of comparative importance stands out prominently, namely, that the earlier and therefore the lower forms of animal life are nearsighted or myopic; and that farsightedness or hyperopia begins to appear as a visual trait in the higher orders which include birds, quadrupeds, simiæ and man.

In his “Treatment of Shortsight,” speaking of myopic and hyperopic animals, Professor Hirschberg says: “To the first group belongs not only the multiple faceted eyes of insects and crustacea which, by means of careful investigation have been shown to form images of extremely near objects, but also the dioptric eyes of fishes and frogs. A fish's eye in water is myopic; moreover, farsight would be useless to it, since at no great depth the clearest water is impenetrable to light. The frog does not stare at Sirius but snaps at flies which venture within its range.

“The birds and large mammals belong to the second group, namely, that of the farsighted animals. The first mentioned being very farsighted while the latter are less so to a considerable degree. Whenever on examining a horse's eye by means of an ophthalmos-

scope, I discovered a marked degree of myopia, a diseased alteration of the eyeball was invariably found to be the cause—in most cases the commencement of senile cataract."

Another fact of the utmost importance in connection with the evolution of vision is the change in the nervous structure. While no distinct allusions were made, the implied understanding is that every external modification has a corresponding inner neural change, invariably associated with it. The difference between the external forms of an *ascidian* and an *Einstein* for instance, necessarily involves a proportionate difference in their respective nervous equipments—equipments which give one a sentiency a shade higher than mere chemical action and furnish a mind to the other which thinks in terms of stellar systems.

And what is, in this case, true of the organism as a whole is equally true of its separate organs. The contrast between vague sensitiveness to light waves marking initial vision and clear image perception characterizing developed vision, is not greater than their respective nervous structures—structures which, in the former, are represented by a solitary peripheral perceptive cell connected with a central neurone by a slender fiber; and in the latter, by an elaborate network of perceptive elements, whose axis cylinder extensions, numbering as high as a half million, bring about relations and associations with different centers of the most involved order.

Passing over the gradual synthesis of the nervous elements in the evolution of the eye, such as the formation of ocelli, rhabdoma and the like, there are a few leading features connected with this process which have to be considered. Their importance for this analysis lies in the fact that they serve as guides in the study of that which concerns us most—*human binocular vision*.

The first of these is the change from a complete into an incomplete chiasmic decussation. This process of reduction which begins in the lower mammalia reaches its height in man where the decussation is reduced to a half or semi-decussation. But this increasing reduction in the number of decussating fibers which is observed as the evolutionary scale is mounted goes hand in hand with a similar reduction in the angle which the optic axes form with the median line. This correlation may be grouped under three heads:

1. Cases of complete decussation. Reduction = 0. *None* of the fibers go directly from optic nerve to optic tract of the same side. Eyes are laterally placed. Optic axes, if produced, form a straight line. Angle with median line = 90° .

2. Cases of semi-decussation. Reduction = 50 per cent. *Half*

of fibers go directly from optic nerve to optic tract of the same side. Eyes are situated frontward. Optic axes are parallel to median line and therefore parallel to each other. Medio—lateral angle = 0° .

3. Cases of partial but not semi-decussation. Reduction = $(50-X)$ per cent. Some of fibers go directly from optic nerve to optic tract of the same side. Optic axes are divergent. Medio—lateral angle = $(90-X)^{\circ}$.

From the foregoing it will be noticed that the reduction in the decussation, though it is simultaneous with that of the angle, proceeds at a different rate, since the highest reduction in the decussation is only 50 per cent, while the corresponding reduction in the angle is 100 per cent. In other words, for every unit of reduction in the decussation there is a two unit reduction in the angle. In the case of the horse, for example,³ where the nondecussating fibers amount to one-sixth of the total, the corresponding angular reduction will equal one-third of a right angle or 30° .

This static or anatomic feature of semi-decussation is associated with two active phases: one pathologic, the other physiologic.

The pathologic phase is demonstrated in cases where the nervous structures concerned in the act of vision are affected on one side. The site of the lesion may be anywhere along the line from the point of decussation to their distal terminations in the visual area of the cerebral cortex. All the classical bilateral anopsias of different geometric forms are then observed.

The physiologic phase is manifested in the simultaneous transmission to the higher brain centers of two similar images impressed upon symmetrical portions of the retinae. These images instead of being seen separately are, by virtue of a cerebral faculty, fused, and we become cognizant, in consequence, of only one image.

In what way this fusion is distinctive and different from that in the lower orders, will be readily appreciated by examining and comparing the two processes effecting fusion: approximation and superimposition.

Approximation, or the *fusion of the two fields* of vision, is limited to group one, where the optic axes, if produced, form a straight line; and where each eye, in consequence, is having a different image. In these cases, vision is lateral and the bringing together of the respective fields is all that is necessary. The visual area, instead of being interrupted by a gap or a dark sector, is by approximation rendered continuous.

³ According to Obersteiner.

Superimposition, or the *fusion of the two images*, takes place in group two where the optic axes are parallel and where both eyes, as a result, are having the same image. Under these conditions vision is frontal and the blending process is absolutely essential. The object fixed, instead of appearing double, is by superimposition perceived as one.

Superimposition literally means overlaying; mere overlaying, however, regardless of specific positions, would as readily *confuse* as fuse. To have superimposition invariably associated with perfect fusion it is indispensable that the parts superimposed correspond throughout with respect to their "points of identity." But an image thus formed must be stratified or laminated—must occupy two planes instead of one; hence fusion by superimposition (the kind occurring in human binocular vision) inevitably leads to tridimensional or perspective vision.²

Experimental verification is afforded by the stereoscope. Two pictures of the same design (the best for this purpose) are pasted or

² But why must superimposition of the projected images result in their occupation of successive planes? Should not images, which are not material things, *so* fuse as to "occupy the *same* space at the same time"? The answer to these questions is found in a further analysis of the involved process. Obviously, to have fusion occur in the same plane it is rigidly necessary that the refractive condition of the two eyes be *exactly* the same; that the plane of *clearest* focusing be *absolutely* equidistant from the nodal points of the respective eyes. But such a condition is rarely, if ever, found in nature. Absolute isometropia, like any other absolute equality, is an abstract conception, not a concrete fact; an hypothesis, not a reality. (See J. S. Mill's "System of Logic," First Part, "Demonstration and necessary truths.") The consequence is that *anisometropia*, or a *difference* in the refractive quantities of the eyes is the universal rule; and this means that, binocularly, clearest focusing is established in *successive* planes. But—it may be further argued—is it not likely that the difference in the static refraction of the two eyes is overcome or taken care of by a *dynamic* element?—that during the act of fixation, either an increased accommodative effort in one eye, or a relatively lessened effort in the other, evens up the inequality and thus brings about distinct image perception in the *same* plane? But the answer is again "No!" Every accommodative effort implies the transmission of an efferent nervous impulse to the ciliary apparatus. Such an impulse, however, can not produce "X" D. of accommodation in one eye, and "Y" D. in the other. It cannot do it because the impulse is not only conveyed bilaterally but also simultaneously and coequally—stimulating both eyes at the same time and to the same *degree*. Note the facts in paralytic squint. The unusual stimulation imparted to the affected eye when made to fix in the field of action of the paralyzed muscle, is reflected synchronously by a greater "secondary deviation" in the screened healthy eye. The case is therefore one, mathematically speaking, of "equals added to unequals"; and though the resulting sums are, by such an operation, rendered less unequal than before, yet the addition of such equals even if carried on *ad infinitum* will never result in absolute equality. Hence it follows that in all, or almost all, cases of binocular vision, clearest focusing occurs in *successive* planes.

Will it be rash to generalize that since anisometropia and stereopsis are a twin feature of frontal vision, that the first is the determining factor of the second?

printed on an oblong card. As such they are, or each one separately is, two dimensional—flat. But when the same card is looked upon with both eyes at the same time through the two glasses of the instrument perspective appears. With the fusion of the two pictures into one there is an immediate, almost magical change from a plane into a solid figure. The question naturally arises, how does it happen? Whence this sudden transformation? The answer is that the “windows” through which the eyes are looking are provided with prisms base out; and prisms base out displace the image inwardly towards the nose. As soon, therefore, as the images are, by the displacement, pushed towards one another, they become superimposed—begin to occupy successive planes and this, as already pointed out, develops subjectively perspective relief.

The evolutionary process accordingly accomplished its utmost along optical lines, when binocular vision attained its relatively high grade of perfection in man.

But what are the characteristics of this binocular vision and what makes it different from the so-called monocular vision? The etymologic derivation gives no hint to the physiologic significance of the term. For while it is true that every being who has binocular vision must have two eyes to see with, yet the converse does not necessarily follow. A hare, for instance, has two eyes, but his vision is, nevertheless, monocular. The key to the solution of this interesting problem is only found in the study of comparative ophthalmology. “For”—as the classics say—“to know a thing means not only to group it with the like, but to separate it from the unlike,” and so, in this case, to understand human binocular vision, it is necessary to contrast it with the other vision which is not binocular but monocular; which is not perspective but panoramic and which is not stereoscopic but periscopic. The next step, therefore, in the further study of the subject, is an analysis of vision in general.

Vision is either direct or indirect. An object is said to be “fixed” or “sighted,” when the eyes look directly at it, or the gaze is concentrated upon it. The image, under these conditions, is formed on the central or macular portion of the retina. Hence, such a vision is also called central or macular vision. Associated with, but terminologically differentiated from the direct is the vision called indirect.

When an object is “fixed” or seen directly, the field of vision is not limited by the boundaries of the object. On the contrary, with the distinct, direct vision of the object which is situated in the center of the field, there is a simultaneous perception of a visual area surrounding it in the form of a circle and extending far out in all directions.

Truly, the object fixed is seen best, most distinctly, while the surrounding peripheral area is perceived less vividly; yet as a component in visual appreciation, the two are completely fused: one insensibly shades into the other. So that ordinarily, and before attention is called, the existence of two separate fields is not even suspected. Only by mental abstraction, or by mechanical limitation as by looking through a gun barrel, do we become conscious of and recognize the fact that fixation is possible by complete exclusion of the peripheral field. And because objects in the periphery of the field, are seen indirectly and the part of the retina affected is removed from the center, is the vision called indirect or eccentric vision.

Exclusively central vision is found only in pathologic cases, where the periphery of the eye is not participating in the visual act. The most characteristic field of pure central vision is seen in advanced stages of retinitis pigmentosa and congenital hemeralopia. In such instances the degenerative changes affect the entire retina with the exception only of the central or macular portion; and the unfortunates afflicted with this dreadful malady have exclusive tubular vision: see only what is in front of them, but nothing around. In the day time they somehow manage to get along, but at night or in the dark, when general or peripheral vision is of the utmost importance to determine the whereabouts, they are, for all practical purposes, completely blind. Hence the name night blindness or day vision (hemeralopia). And because night vision (nyctalopia) draws principally upon the circumferential elements of the retina, their full exposure by an associated dilation of the pupil (mydriasis) is absolutely essential. All those, therefore, who use night vision only (owl, bat, underground rodentia) or mainly (prowling, nocturnal carnivora) have unusually large pupils. In some of the latter their mydriatic condition is betrayed by an uncanny phosphorescent-like glare; which is nothing more than a reflection from the interior of the eye, of the ordinary night light, by a choroidal membrane called tapetum lucidum. Accordingly, cortical cataract or anything which binds the iris down and prevents its dilatation, as an annular adhesion for instance—so-called seclusion of the pupil—will, by shutting off the peripheral retinal elements, bring about a condition of night blindness.

In contrast to night vision with dilated pupil, stands day vision with contracted pupil. Day or direct vision, it will be remembered, is accomplished mainly, and at times exclusively, by the exploitation of the macular elements—elements which are distinguished from the peripheral ones by their power to form clear and distinct images. But with the pupil dilated, a superfluous number of rays enter the

eye at the periphery and produce diffusion circles or blurred images—a condition technically known as spherical aberration. To prevent this undesirable intrusion, therefore, and to preserve great distinctness of images, contraction of the pupil by the diaphragmatic action of the sphincters, is absolutely necessary. In brief, hemeralopia is associated with miosis, while nyctalopia goes along with mydriasis.

An histologic difference in the perceptive retinal elements which respond to shady vision (scotopia) and full light vision (photopia) respectively, remains to be pointed out.

It has been found that those animals which see only or clearest in the dusk and see nothing or very little in daylight, have no cones in the retinae. The only elements present are the rods; while those who see most distinctly in daylight have cones in addition. Nay—the part best adapted for full daylight vision—the macula—*has no rods at all*. This intimate association of nocturnal vision with rods on one hand and of diurnal vision with cones on the other, call forth some interesting speculations concerning the specific nature of the reaction of these retinal elements.

The causal relation between the act of seeing and the bleaching of the visual purple is hardly questioned by anyone. Still less questioned is the fact that this photochemical process can go on only so long as the capillary loops of the choroid restore the necessary pigment elements as fast as they are used. But this constant renewal of the visual purple by the uveal circulation—say some—is only possible where the drain upon this substance is comparatively limited, as at night for instance, when the rays entering the eye are relatively few and widely interspaced. In that case—the argument proceeds—supply and demand are evenly or ideally balanced, and vision, everything else remaining the same, can go on, so to say, “ad infinitum.”

A different aspect, however, is presented by daylight and day vision. In this case, owing to the fact that the rays are more closely set or the beams more compact, the photochemical activity would be very intense and the visual purple would exhaust rapidly. Sight, instead of being continuous would, under these conditions, be rhythmically interrupted—interrupted until the consumed elements are replenished once more. Nature, therefore, to avert this, utilizes physical instead of chemical processes for the transmission of visual stimuli. The protoplasm within the cone cell, in response to light, contracts, and this contraction imparts a fine mechanical impact to the nerve end connected with it, and an impulse is thus conveyed along the axis cylinder to the visual area of the cortex. In support of this idea, the fact is adduced that each individual cone cell is, unlike the rods, in direct relation with a separate neural extension. The sum

total of the above reasoning amounts to this, that while night vision is photochemical in reaction and the elements engaged are the rods, day vision on the other hand is phototropical in reaction and the structures responding are the cones.

May it not be possible, that inasmuch as night vision is monochromatic and day vision polychromatic, that the cone cell is the seat of color discrimination?

These are some of the differential traits distinguishing direct from indirect vision. Additional ones will be mentioned before this discussion is ended. The main fact, however, to which all the others are tributary is that direct binocular vision developed from indirect monocular vision by a slow and gradual process—that the entire phylogenetic series of vertebrates, beginning with the amphioxus and ending with man, represents this evolutionary transition.

The principal change which initiated and brought to a conclusion this progressive process, is *the migration of the eyes from their original lateral habitat—frontward*. The immediate effect of such a change is the overlapping of the fields of vision. Primarily separate or at best contiguous, the fields of vision encroached upon each other farther and farther in proportion as the eyes, in their march to the front, took up positions more and more advanced. The limit was reached when the optic axes became parallel. In man this overlapping amounts to 60° on either side of the median line, that is, the right eye sees the right field of vision plus 60° of the left; and the left eye sees the left field of vision plus 60° of the right (Worth).⁴ In other words, while the extremes of the fields are seen by each eye separately, indirectly or monocularly, the central or overlapped portion is seen with both eyes jointly, directly or binocularly.

The physiologic aspect of this new situation is, as already mentioned, that with the simultaneous exposure of symmetrical portions of the retinae, during the act of fixation, two similar images are formed which are welded by a process of superimposition and perceived as one. The importance of this feature—of having corresponding retinal areas affected in the act of direct vision, is demonstrated in cases of paralytic squint. Owing to the fact that in these instances the strabismic eye is drawn away from parallelism by the active antagonist, the retinal parts receiving the images are noncorresponding or asymmetrical and the consequent vision is double.

⁴ The *exact* numerical expression as given in the original text, is as follows:

“When the eyes are in the primary position the fields of vision of the two eyes overlap everywhere, except in a sector of about 35° towards the temporal periphery of each field.” (Worth. Squint., London, 1921, p. 7.)

The absence of diplopia in ordinary cross eyes or concomitant squint is not an instance showing exception to the rule, but one lending supportive evidence and proving its absolute invariability. Whether squint is due to muscular imbalance, to refractive anisometropia, or, as Worth argues, to a defective or slowly developing fusion faculty, the fact remains that in the early stages of concomitant squint *there is diplopia*. Gradually, however, owing to "mandates" issued from the vision center, the false image is being systematically suppressed until the squinting eye is thrown out completely from participation in the visual act; and for all practical purposes is rendered blind from disuse—"amblyopia ex anopsia." When asymmetrical or noncorresponding parts of the retinae are affected in the act of fixation, the results are either diplopia or monocular vision. In the former instance two images are taken cognizance of; in the latter, one. The image of the eccentrically placed eye is suppressed.

The shape of the lens also undergoes a decided change, as macular vision is taking the place of peripheral vision.

In the lower *mammalia* where vision is indirect or circumferential and where therefore clear focusing on *any* part of the retina is essential, the lenses are spherical. Man and the allied species belonging to group two, on the other hand, whose vision is almost exclusively direct or circumscribed and who, therefore, require distinct image formation only on *one* spot—the macula—have their lenses flattened in the sagittal diameter. To counteract the loss of refractive power of the lens as it passes from a spherical into a flattened shape, the corneal curvature has a relatively shorter radius and the vitreous chamber is proportionately lengthened—changes which produce a compensating condition of curvature and axial myopia.

How the spherical form is gradually departed from as the zoologic type advances, is given in "lentic metric" tables by Treacher Collins in his "Arboreal life and the evolution of the human eye."

On examining those tables, it will be found that the successive alterations in the shape of the lens and the consequent changes in the proportion of its diameters, indicate, in arithmetical terms, the comparative progress any given eye has made in its development from diffuse to concentrated vision.

Hand in hand with the transition of panoramic or periscopic vision into perspective or stereoscopic vision is the decrease in the area of the cornea as compared with the length of the eye ball.

Everything else remaining the same, the interests of panoramic vision are served best by a relative increase in the area of the transparent surface. The visual fields being of greater dimensions,

peripheral images are more readily discerned. Conversely, perspective vision, which is characterized by vivid central and faint peripheral images, is aided considerably by a restricted exposure.

Deduction is verified by induction. The numerical ratios obtained between the horizontal lengths of the corneae and the antero-posterior diameters of the eyeballs, in various animals, as recorded by Collins, point indubitably to a mathematically graded scale of relations between the type of vision and extent of transparent area.

The figures, expressed in mm., are as follows: in man, who has the highest perspective vision, the ratio is 11:24, less therefore than a half; in the cow, horse, sheep and pig, who have partly panoramic and partly perspective vision (group three), the relation, in successive order is 22:18, 44:34, 27:19, 23:17—more than a half—almost two-thirds; and in the rabbit, rat and hedgehog, whose vision is panoramic, the ratios are 16:15, 5.5:5, 6.0:6.0—the same or nearly the same. The absence of central vision in some of these rodents is at times so striking that "as all sportsmen know, rabbits and hares see very imperfectly when danger is immediately in front of them and will run straight forward toward a gun" (Collins).

The following summary may here be fitly introduced as a recapitulation of traits differentiating the two kinds of vision:

VISION

<i>Direct</i>	<i>Indirect</i>
Frontal	Lateral
Binocular	Monocular
Perspective	Panoramic
Stereoscopic	Periscopic
Central	Peripheral
Distinct	Faint
Circumscribed	Circumferential
Photopic	Scotopic
Phototropic	Photochemic
Polychromatic	Monochromatic
Miotic	Mydriatic ⁵
Hemeralopic	Nyctalopic

⁵ An interesting fact, corroborative of the relation between lateral monocular vision and mydriasis is the so-called "Tournay's-Reaction."

"When a man whose ocular apparatus is normal, whose pupils are equal, reacting normally to light, contracting normally and equally with movements of convergence and accommodation, looks strongly to his right and maintains this position, the right pupil becomes larger than the left. Thus isocoria being the rule in anterior fixation, anisocoria becomes the rule in lateral fixation." Doyne, Br. J. Oph., 1923.)

It is desirable, however, that the contrasting terms in the respective columns, are not to be interpreted in a strict or literal sense, but rather in a liberal or philosophic sense. With the exception of those special instances where vision is exclusively of one kind, either central or peripheral, the vast majority of cases have both kinds: direct and indirect—a *mixed* vision. The question is only which one predominates. In man, for instance, it is principally direct; in the lower vertebrates it is almost entirely indirect; while the intermediate forms have more or less of a “fifty-fifty” proposition. The information, therefore, which the above antonyms intend to convey is, that the nearer vision approaches either extreme, the more it assumes the characteristic feature of that specific type. The more eccentric, for example, man’s vision is the more lateral it is, the more faint, the more scotopic, the more monochromatic, etc.; and *vice versa*. And what is true in the above case where the two kinds of vision coexist, is equally true where they are separate.

Have the facts so far adduced proven conclusively the object of the discussion—that direct vision evolved from indirect vision? The answer is: implicitly, yes; explicitly, no. While the general understanding is that the lower orders are the earlier and therefore by antedating chronologically the higher have given rise to them, yet no specific statement directly linking the different orders as successively *caused forms* has been made. This, as a conclusion, is now supplied.

It is known that the series of vertebrates, geologically, runs in the following order: fishes, reptiles, mammals and primates. In other words, the time was when fishes were the only vertebrated beings on our planet. Later, however, reptiles developed; and while they haven’t completely supplanted their progenitors, they have nevertheless become the dominating animals; they have occupied the seas, as swimming reptiles; the air, as flying reptiles, and the terra firma as walking reptiles. The next change in the fauna was when the mammalia came forth. At first aplacental, eventually placental. The mammalian hordes have in their turn taken over from the reptiles the commanding position on the earth; and the former prehistoric saura have dwindled and degenerated into the present surviving types. Finally man appeared and assumed control of earthly affairs. Now, when these data are coupled with others such as that fishes and reptiles have their optic axes, more or less, in a straight line; that the lower mammalia have them divergent and that man has them parallel—if this is done, the transition of direct into indirect vision becomes an absolutely demonstrable fact.

Embryologically, the above truth is attested in a similar manner. The angular relation between the planes of the primary optic vesicle and the fully developed eye (in the human embryo) is in complete accord with the foregoing interpretation. If ontogeny, the "minute hand" of evolution, retraces the path of phylogeny, its "hour hand," upon the dial of biologic time; if the successive *changes* manifested by the individual in utero, portray the successive *forms* of his lineal ancestry—then the evolution of frontal from lateral vision is a matter of *direct observation*. The geometric collocation of the primary optic vesicle with the eye of the lower vertebrates, points conclusively to the genetic relationship subsisting between the two. And it also comes to mean that the consecutive points of the curve along which the developing eye is moving to reach the *anterior* plane, represent the successive "stations" on the arc of a quadrant, passed through by the ancestral eye in *its long migration from the original lateral habitat—frontwards*.

SENSORY DISSOCIATION IN SPINAL CORD LESIONS
WITH NOTES ON SENSORY-PSYCHIC INTEGRATION

BY JOSEPH BYRNE, A.M., M.D., LL.D.

DEPARTMENT OF BIOLOGY, FORDHAM UNIVERSITY; ATTENDING NEUROLOGIST,
CENTRAL NEUROLOGICAL HOSPITAL; CONSULTING NEUROLOGIST, FORDHAM
DIVISION, BELLEVUE AND ALLIED HOSPITALS, NEW YORK CITY

(Continued from page 473)

CASE V. INTRAMEDULLARY LESIONS (MULTIPLE SCLEROSIS) CAUSING: (1) PROTOPATHIC DISSOCIATION OF SUPERFICIAL AND DEEP VARIETIES; (2) REMISSION PERIODS IN WHICH AS SUPERFICIAL CRITICAL SENSIBILITY BECAME RESTORED SUPERFICIAL AFFECTIVE LOST ITS PROTOPATHIC CHARACTERISTICS, THE DEEP CRITICAL AND AFFECTIVE FORMS REMAINING UNAFFECTED.

S. B., thirty-five years. At thirty-three and a half years began to lose power in left leg; with this the left leg, about and below the knee, behaved as if it were "asleep." Left leg and foot extremely sensitive to "touch" and to hot and cold liquids. At times pains and a burning, sticking feeling about right knee. Mentality good; subject to emotional outbursts. The sensory condition is set forth in Chart 8. The status of the left foot and lower portion of the left leg as found upon examination before, during and after a remission period is herewith set forth in summary.

	Before	During	After
Superficial critical	0	+	0
Superficial affective:			
Hurt element	+++	+	+++
Specific critical elements	+ ♀ ♀	+	+ ♀ ♀
Protopathic characteristics	+	0	+
Deep critical	0	0	0
Deep affective:			
Hurt element	+++	+++	+++
Specific critical elements	0 or ♀ ♀	0 or ♀ ♀	0 or ♀ ♀
Protopathic characteristics	+	+	+

DISCUSSION

The marked impairment of deep critical sensibility ("astereognosis," etc.) in the right hand of Case 1 points to a cortical sensory lesion on the left side received when the patient's skull was fractured. The ptosis enophthalmos) and smaller pupil of the right eye, as well as the absence of sweating on the right side of the head and face, in Case 2, points unmistakably to compression of the efferent cervical sympathetic paths somewhere within the cord at or cephalad of their

exit through the upper thoracic ventral roots. In both of these cases the development of the lesions responsible for the sensory dissociations seemed to bear some relationship to the injuries received at an earlier date. Compare the mechanism of cerebral maldevelopment following cranial injuries received during childbirth, etc., and the etiological relation of head injuries to the development of brain tumors.(25) In Case 1 the onset of the symptoms soon after the decompressive operation is significant and should make surgeons

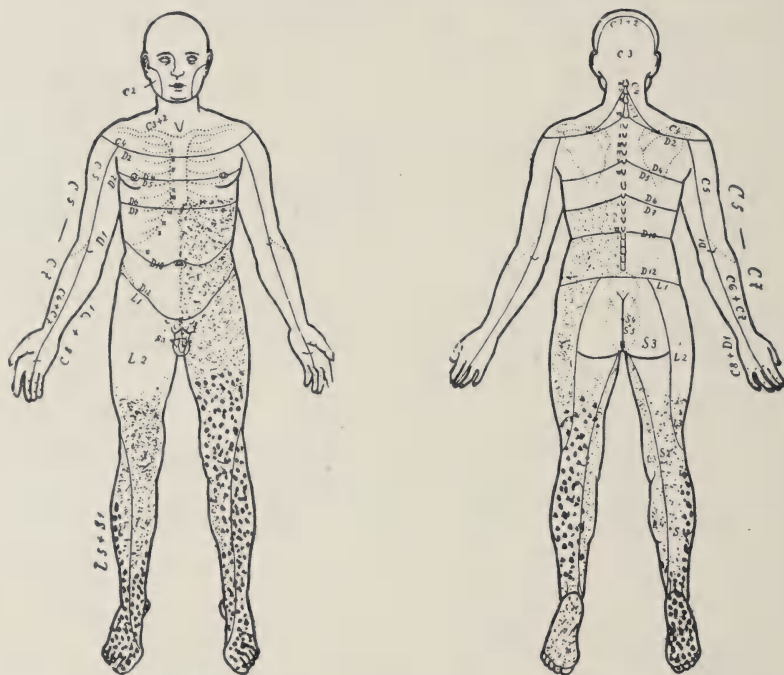


CHART 8. Case 5. Disseminated sclerosis. Protopathic dissociation. *Heavy dots*: lost sensibility for cotton and the 0.5 gramme von Frey hair. *Light stippling*: impairment for cotton and the 0.5 gramme von Frey hair. Superficial affective sensibility retained, with marked protopathic characteristics, on left thigh, leg, and foot, and about right knee.

give due weight to all possible contra-indications before undertaking the apparently harmless decompressive operation.

Since the publication of the clinical work done by Schultze,(2) and the later clinical work by Kahler,(23) and by Schlesinger,(24) lesions of the syringomyelic type have been a fruitful field for investigation of the sensory pathways in the spinal cord. In such conditions, however, only one form of dissociation has hitherto been established as the result of the spinal lesion. In Cases 1 and 2, two

forms of sensory dissociation are discernible (1) *critical* due to direct destructive effects in the central gray matter and (2) *protopathic* due to indirect effects of the lesion viz. compression of the long tracts on the left side of the cord in Case 1 and on the right side in Case 2. In its indirect effects the lesion behaved like an extramedullary tumor compressing preponderately one lateral half of the cord thereby inducing the Brown-Séquard syndrome. In such conditions the over-reaction, which ordinarily serves as the main index of protopathic dissociation, may itself become dampered at an early stage owing to partial conduction block in the lateral spinal lemniscus (spinothalamic tract). In the critical dissociation the outstanding feature was the suppression of the gross affective (hurt) element with preservation of the specific critical elements for such complex forms of stimulation as pricking, "heat" and cold. This corroborates the author's findings in a previous contribution (1) in which was demonstrated the existence in the peripheral nerves of separate sets of pathways for the mediation of the gross affective (hurt) element as opposed to the critical elements. Noteworthy, however, is the fact that the specific critical elements did not entirely escape. Thus in Case 1 on the left chest the warmth element was suppressed for all degrees below 45° C. and preserved, without the element of hurt, for 45° to 70° C., the specific element "cold" being absent for 20° to 0° C. on area bounded by heavy dots in Chart 2, and the specific element "pointedness" preserved everywhere for pricking at 3.0 to 12.0 grammes. In Case 2 the specific element warmth was practically unimpaired, whereas the specific element cold was absent for 22° to 0° C. upon an area coextensive with area of hurt-loss for pricking at 3.0 to 27.0 grammes. These facts show that the specific element in each of the three superficial affective stimuli, viz., pricking, "heat" and cold is mediated by a separate set of pathways each of which is capable of being functionally interrupted independently of the others. The facts further show that the pathways lying next to the central canal mediate the gross affective (hurt) element which may be regarded as the primary basic element in affective stimulation. The paths mediating the specific element "cold" lie next in order from within outward to those mediating the gross affective (hurt) element. As the deep affective is the last form to disappear in cord compression from without (Case 4), it seems that cold must be regarded as a more resistant (older) form than warmth for this latter type of suppression. This finds corroboration in the fact that after section and suture of a peripheral sensory nerve this element recovers somewhat in advance of warmth. The paths for pointedness of pricking

cross in the central gray matter in a position still farther removed from the central canal since this was the specific element least affected in Cases 1 and 2. The absence or marked impairment of vibration, which may be regarded as summed pressure-touch, in Cases 1 and 2 for both the critical (rate) and affective element (quality) points to this as one of the very oldest forms. Compare the recovery (after complete abolition) of this form in Case 3 on the second day following removal of the tumor that blocked conduction in the long tracts.

In Case 4 the augmentary influence of the labyrinthine mechanisms (otoliths) upon the mechanisms of postural tonus was remarkably in evidence on December 19 and 21, 1916, the abolished knee jerks reappearing when the patient was placed sitting on the side of the bed. Compare the work of Magnus and De Kleijn on the function of the otoliths.(3)

The level and vertical extent of the lesion in Cases 3 and 4 point to compression of the long tracts as the sole factor in causing the protopathic dissociation found in the lower limbs. Thus in Case 4 the cord compression abolished sensibility in order as follows: superficial critical, deep critical, superficial affective, deep affective. As indicated in a previous paper (4) the cord compression from without gradually suppressed function first in the long tracts mediating the superficial critical forms because these represent more highly specialized and hence more vulnerable forms for this type of injury than the deep critical forms. Besides being more resistant the tract (lateral spinal lemniscus) mediating the hurt element in both superficial and deep affective sensibility, because of its more central location within the cord, is more protected from the effects of cord compression from without than the tracts (fasciculus gracilis et cuneatus and ventral spinal lemniscus) mediating the critical forms. Hence the affective (hurt) element became involved later than the critical elements—the more resistant deep affective element later than the superficial affective. The tract mediating the affective (hurt) element does not, however, even in the early stages of compression from without, entirely escape. Indeed compression of the long tract mediating the hurt element (lateral spinal lemniscus, spino-thalamic tract) is itself a factor in the production of the phenomena (over-reaction, etc.) which characterize protopathic dissociation, a secondary result of such compression being dampering (early stage) and final suppression of these very characteristics. In Case 3 the order of return of sensibility for the various forms confirms the order of disappearance observed in Case 4 and both cases show the relation

which the abolition or restoration of critical sensibility bears to the induction or suppression respectively of protopathic characteristics, superficial critical sensibility manifestly exerting an inhibitory influence upon superficial affective, and deep critical upon deep affective. In Case 5 this influence was apparent in both phases, *i.e.*, during the abolition and restoration of superficial critical sensibility.

Order of Return of Sensibility After Long Tract Compression. In Case 3 the order of return of sensibility is noteworthy. On the second day it was found that the deep affective (hurt) element had become markedly restored. This represented improvement in the one form that had just escaped abolition before operation. The return of vibration coincident with improvement for the deep affective (hurt) element indicates that this form is practically as resistant as the deep affective (hurt) element itself. And as the critical element of this stimulus (vibration) also began to be restored on this day it seems that one of the deep critical forms, *viz.*, pressure-touch, is practically coeval with the oldest and most resistant (to external compression) of all forms, *viz.*, the deep affective (hurt) element. It seems, therefore, that as between the fundamental critical element (touch) and the gross affective (hurt) element it cannot be said that as regards origin or time of functional acquisition either is prior to the other. Compare the absence or impairment of vibration in association with absence of the hurt element in Cases 1 and 2. Compare, further, Byrne.(1) The return on the fifth day of the deep critical elements, and the coincidental recession of the protopathic characteristics associated with deep affective stimulation were significant as showing the inhibitory control exerted by the deep critical forms as a *whole* upon the fundamental deep affective (hurt) element. The return on the 5th day of the superficial affective (hurt) element, the superficial critical elements remaining suppressed, showed once again the superficial affective system in operation uncontrolled by the superficial critical system and manifesting protopathic characteristics. The partial return on the tenth day of superficial critical sensibility coincided with partial recession of the protopathic characteristics attending superficial affective stimulation. Here is evidence of inhibitory influence exerted *fractionally* by the superficial critical upon the superficial affective system. The functional antagonism, total and fractional, that obtains between the critical and affective systems is a factor of fundamental significance in sensory-psychic integration. In normal individuals, however, the control exerted by the critical upon the affective system is limited. When the noxious element reaches a certain degree of intensity the critical control is

over-ridden and the individual moves away or withdraws the part stimulated or does both. The affective system, within certain limits, is thus controlled as a whole by the inhibiting influence exerted by the critical system as a whole. In addition to this general inhibition, in each particular act of stimulation, *e.g.*, by moderate pin-prick, or 40° to 45° C., the particular critical mechanisms activated inhibit the particularly related affective mechanisms. This also is a fact of fundamental significance in sensory-psyche integration.

Root versus Tract Pressure.

It might be questioned whether the protopathic characteristics observed in Cases 3 and 4 were not the result of root-, instead of tract-pressure. The location of the neoplasm in each case, however, manifestly points to long tract compression as the cause. In Case 4, moreover, the persistence of the spinal reflexes in the lower limbs, after abolition of all elicited forms of conscious sensation, is conclusive proof that the afferent and efferent arcs, *e.g.*, of the nociceptive reflex, were patent in the nerve roots whilst the related pain paths passing brainward were completely obstructed at a higher level.

Fallacy of Tract Irritation

Spontaneous pains resembling the pain crises of tabes appeared in Case 3 on the ninth day. Present day neurologists would attribute such pains as these to "tract irritation" induced by pressure of the neoplasm. The word "irritation" as used by clinicians is a hang-over of a bygone day that reminds one of the pains unaccompanied by definite local signs which were so frequently diagnosed as "sympathetic" before John Hilton (26) demonstrated that all such pains were the result of lesions of the nervous system often far removed from the site to which the pain was referred. Compare Byrne, "The Mechanism of Referred Pain, etc." (5). To the author tract irritation lacks finality and bears little relevancy unless backed by some rational explanation which would account for the pains without conflicting with well established facts. Nerve tracts normally, and indeed the spinal cord as a whole, are *par excellence* conducting mechanisms and not mechanisms for the *initiation* of nerve impulses. If the latter were the case it would tend to defeat the very purpose the nervous system serves in the mechanisms of protection and ordered sensation. It is well known that nerve fibers are relatively insensitive compared with their receptor mechanisms. In Case 3 the absence of pain during the period immediately preceding operation cannot well be explained on the theory of "tract irritation" since the pain

tract undoubtedly continued to be "irritated" at the cephalic limit of the neoplasm even though one were to admit that, at the focus of compression, the lesion had passed from the "irritative" to the "destructive" stage. That this latter was not the case, anatomically at least, is shown by the survival of the deep affective (hurt) element before operation and by the rapid, almost complete restoration of all forms of sensibility after relief of the cord compression. Something more than "irritation" of mere conducting paths, whether in the spinal cord or in the nerves, or nerve roots, must be invoked to account satisfactorily for the pains of tabes as well as those that occurred in Case 3. Compare Byrne, "The Mechanism of the Gastric and Pain Crises of Tabes" (6).*

Present Status of the Nature and Mode of Propagation of the Nerve Impulse

Although the nature of the nerve impulse is not yet known, within recent years, thanks to the work of Lucas and Adrian, much light has been thrown on the mode of its origin and propagation (conduction). According to present conceptions the energy represented in the propagated disturbance (conduction) comes primarily from the nerve fiber itself rather than from the stimulus. Lucas (27, p. 23) compares it to the burning of a train of gunpowder. This conception fully accords with the author's views if the fact be kept in mind that the axone is a protoplasmic process continuous, functionally and anatomically, with the protoplasm of the parent neurone body. In experiments (unpublished) done in 1915 by means of interrupted areas somewhat similar to those used by Adrian (28) the author found that after moderate injury of the cervical sympathetic nerve recovery of "irritability" (local excitatory process) and "conduction" (propagated disturbance) occurred more readily when functional continuity with the parent neurone bodies was uninterrupted. In view of the experimental facts it is no longer permissible to consider the nerve impulse merely as an

* From the standpoint of animal experimentation Dusser de Barenne's (39) is perhaps the most important contribution to the study of sensation. In addition to a careful perusal of his contributions the author had a delightful heart to heart discussion of them with Dusser de Barenne himself at Edinburgh in July, 1923. The author believes that at best but little reliance can be placed on sensory interpretation in animals after such lesions as the strychninization method necessarily entails. In any event the evidence so far adduced by the Dutch observer invalidates none of the basic arguments in the author's Theory of the Mechanism of Pain. (4) After section of 6 or 8 posterior nerve roots the fact that the animal "bites at" the skin area supplied by these roots can be accounted for without postulating for the dorsal horn cells any new and unwarranted rôle.

electric current transmitted along the nerve fiber as along an insulated wire. The mechanism in the two modes of transmission is essentially different. Care must be taken to distinguish between the nerve impulse proper and the associated electrical response (action current) which accompanies activation of all excitable tissues (nerves, muscles, glands). This consists of a wave of lowered potential which passes along the nerve simultaneously with the passage of the nerve impulse proper. The "all or nothing" law of response, first established by Marey for the heart, has apparently been well substantiated for nerve by Adrian.(29) It must be borne in mind, however, that electrical stimulation of a nerve bundle is but a crude representation of what takes place in conditions approaching those of normal sensation. In Adrian's experiments the phenomena observed represent at best the effects of an unusual degree of *affective* stimulation, as it is well known that the critical system is with extreme difficulty, or only very inadequately activated by electrical stimulation. In attempting to apply the knowledge obtained in the study of the nerve-muscle preparation to the reflex arc physiologists have ignored the dorsal root ganglia although the neurone bodies in these structures constitute an essential part of the afferent arcs. To the author, it seems that in studying the reflex arc it is essential to take into consideration the status of the neurone bodies in the dorsal root ganglia. The old conception, based on the work of Bethe,(30) Steinach (31) and others, that the nerve impulse normally passes over the T-shaped process from the peripheral to the central axone without entering the neurone body needs revision in view of the present conception of the origin and mode of propagation of the nerve impulse, and more especially in view of the pathological behavior (axone reaction phenomena, hypermetabolism, etc.) of the bodies of the primary sensory neurones after injury of their axone processes as shown in the author's experimental studies.(7) So far the existence or nonexistence of the two main divisions of the afferent nerves, *i.e.*, affective and critical, has not been satisfactorily demonstrated by physiological * experimentation although the findings of Sherrington and Sowton (32, p. 445) points to the existence of two different kinds of afferent fibers in the stimulated nerve. The clinical (1) and histological evidence (33) of the existence of the two great divisions of the afferent nerves is, however, convincing.

* Since this was written the critical and affective systems have been satisfactorily differentiated experimentally (38).

Mechanism of Protopathic Dissociation

The sensory studies of Cases 3, 4 and 5 clearly demonstrate the inhibitory control exerted by the critical upon the affective systems. Removal of this control by preponderate, or absolute suppression of critical sensibility as the result of long tract compression, released the affective systems so that these latter reverted to the more primitive method of functioning according to the "all or nothing" principle, a further factor of significance being hypermetabolism in the primary noci-ceptive neurones, chiefly in the neurone bodies. This hypermetabolism (hyperfunctioning) experimentally established by the author in animals (7) is the biological response of the primary noci-ceptive (gross affective) neurones to injury or conduction block in the primary neurones themselves, or in the related long intramedullary tracts, the object being restoration of anatomical and functional continuity. These are the two basic factors in the mechanism of protopathic dissociation.

Site of Impingement of the Critical Upon the Affective Systems

To the author, in so far as conscious sensation is concerned, the optic thalamus is the lowest level at which this functional impingement takes place. On no other supposition does it seem possible to explain the protopathic dissociation encountered in Cases 3 and 4. Compare the case of Taylor and Beling (8) amongst many others seen by the author, in which a tumor compressed the spinal cord far cephalad of the level of entry of the sensory nerve roots supplying the areas (lower limbs) in which severe spontaneous pains (protopathic dissociation) occurred. If either set of critical pathways came into functional relations with the corresponding set of affective pathways so as to exert its natural control over these *latter*, let us say in the dorsal root ganglia, or in the cord segments near the level of entry of the posterior nerve roots, it is not easy to see how a neoplasm causing cord compression far cephalad of a given set of root-ganglia or cord segments could cause protopathic dissociation, *i.e.*, release from critical control, in peripheral areas supplied by these ganglia or cord segments. Yet this was exactly what happened in Cases 3 and 4. A somewhat similar difficulty is encountered in the protopathic dissociation found in peripheral nerve injuries on the assumption that the critical mechanisms impinge functionally upon the affective mechanisms at the level of the receptors or indeed anywhere in the peripheral nerves. In direct contrast with the results of Head and Thompson (9) the cases studied in this paper show conclusively that the critical paths, superficial and deep, run courses all the way up the cord that are anatomically separate and removed from

the affective paths. Even for a punctiform stimulus such as pin-prick the critical element takes a path different anatomically from that taken by the affective (hurt) element of the same stimulus since in Cases 1 and 2 the latter was suppressed whilst the former remained practically unaffected. Above the thalamus the critical pathways as a whole end preponderately in the outer layers of the cerebral cortex whilst the affective pathways end for the most part in the inner layers of the cerebral cortex. The work of Dejerine and Egger,(10) of Dejerine and Roussy,(11) and of Roussy,(12) corroborated later by the work of Head and Holmes,(13) of Holmes and Head,(14) and of Byrne,(4) has shown that lesions of the ventro-lateral aspect of the thalamus cause marked protopathic dissociation (dysaesthesia of Roussy). To account for this dissociation it is necessary to postulate, as Byrne has done,(4) impingement, by means of collaterals or main-stems, of the critical upon the affective pathways within the thalamus since typical thalamic (protopathic) dissociation does *not occur in lesions of the internal capsule or at other levels above the thalamus*. Compare the prespinal central mechanism of postural tonus in which the afferent critical paths pass to the brain-stem where they directly (not through the cerebral cortex) impinge upon the prespinal center and so exert their inhibitory control upon the lower spinal mechanisms (flexion-reflex, etc.). Compare, also, the frequency and extent to which the deep critical pathways for posture and passive movement, which may be regarded as the psychic adjunct of reflex posture and its variations, are involved in cases which exhibit typical thalamic dissociation phenomena. It seems, therefore, that there is ample justification for the conclusion that the critical does not impinge upon the affective system until the level of the thalamus is reached, and that by means of direct impingement at this level, and not through the cerebral cortex, the main provision is made for the fundamental restrictive control exerted by the critical upon the affective system.

Hypermetabolism and Storage of Neuropotential

The primary noci-ceptive (affective) seem to be the only neurones in the lower sensory mechanisms capable of elaborating and storing a great excess of specific neural energy (neuropotential, kinetoplasm). These neurones, represented as Ranson (33) has shown, in the peripheral nerves by unmyelinated fibers springing from the smaller cells in the dorsal root ganglia are preponderately dynamic in function. Like the secondary and tertiary neurones of all the systems the primary neurones of the critical system, repre-

sented in the peripheral nerves by medullated fibers springing from the larger cells in the dorsal root-ganglia seem to be incapable of marked hypermetabolism in response to injury. The critical and affective systems, as the author has already pointed out,(1) function side by side in a "coöperation of antagonism," each system contributing its elemental quota upon stimulation at the periphery. In normal individuals every stimulus, whether of the critical or affective type, makes appeal, actual or potential, to both systems, preponderately of course to one as against the other. In normal sensation, therefore, pure affective or critical stimulation is a mere theoretical entity. Under ordinary conditions of contact with our environment, as well as in clinical and laboratory studies made upon normal individuals, all stimulation registrable in consciousness carries an affective as well as a critical element although either element may be of minimal degree. To the author this also is a fact of fundamental significance in sensory-psychic integration. In the protopathic dissociation encountered in lesions of the peripheral nerves,(1) brain-stem,(4) or ventro-lateral aspect of the thalamus,(4) owing to abolition of the critical elements, the residual form of sensibility may be preponderately or purely affective in type. On the other hand in lesions of the syringomyelia type and in some peripheral nerve lesions (1) the gross affective (hurt) element may be suppressed, the residual form being purely critical. Compare Cases 1 and 2.

The Sensory Gamut

In the critical system the primary fundamental sensory element is contact (touch), the analogous fundamental in the affective system being the hurt or gross affective element. All the fundamental forms of skin sensibility as such seem to be synthetic derivatives of these two primary elements. In testing superficial sensibility, as ordinarily done at the present time, warmth and cold presuppose contact whilst pointedness is itself an areal modification of contact. For the practical purposes of laboratory and clinical study the ranges of the superficial sensory gamut may be set down thus:

	<i>Higher</i>	<i>Critico-</i>	<i>Affectivo-</i>	
<i>Critical</i>	<i>Critico-Affective</i>	<i>Affective</i>	<i>Critical</i>	<i>Affective</i>
Touch	Touch-point	Point-hurt	Hurt-point	Hurt
Touch	Touch-warm	Warm-hurt	Hurt-warm	Hurt
Touch	Touch-cold	Cold-hurt	Hurt-cold	Hurt

At the extremes of the gamut stand the fundamental elements touch and hurt; but, as neither of these is ever experienced in its

pure form by normal individuals, the actual ranges are represented by the composite forms standing between the extreme fundamental critical and affective elements. In the compound terms the first word (on the left) indicates the preponderant sensory component. For purposes of clinical and laboratory study, more especially from the standpoint of introspective analysis, these terms are superior to the terms in common usage such as "sharpness," which includes touch-point and point-hurt, "heat," which includes warm-hurt and hurt-warm, and "cold," which includes cold-hurt and hurt-cold.

For the most part the superficial sensory forms tabulated in the gamut represent more refined specialization of function than the corresponding related forms in deep sensibility, the ranges of whose gamut are fundamentally identical with those in the gamut of superficial sensibility if we take "touch" as representing pressure-touch and "point" as representing the areal extent of the stimulus as felt. Compare the direct tests made by Byrne (4, 25) on the deep sensory mechanisms.

THE FUNDAMENTAL VARIABLES

In superficial sensibility the critico-affective forms touch-point, touch-warm, touch-cold must be regarded, theoretically at least, as representing respectively the primary fundamental critical forms of sensibility, viz., touch, warmth, and cold, each of which, mediated by its own separate set or system of pathways, and served by its own set of receptor mechanisms, functions in a "coöperation of antagonism" with the fundamental affective element (hurt) mediated by its own special system of pathways and served by its own receptor mechanisms. Compare the known physiological receptor mechanisms, viz., the *touch spots* of von Frey (15) who was perhaps antedated in their discovery by Hensen, (16) and the *warm* and *cold spots* of Blix, (17) who was perhaps antedated in their discovery by Goldscheider, (18) The pain spots are ubiquitous. Blix (22) questions their existence as such although von Frey (15) accepts it. Rivers and Head (20) regard the "heat" and cold spots as receptor mechanisms for their "protopathic forms" of heat and cold. The warm and cold spots are, however, fundamentally critical mechanisms especially in relation to the mechanisms mediating the gross affective element (hurt). In the sensory gamut, therefore, the four primary forms, touch-point, touch-warm, touch-cold, and hurt, correspond to the "fundamental variables," touch, heat, cold, and pain of the older psychologists. These variables may be accepted with the qualification that three of them, touch, warmth, and cold, function in coöp-

eration with the fourth (the fundamental hurt element) in forming the synthetic resultants which constitute all our ordinary general sensory forms.

FUNCTIONAL SPECIFICITY OF THE SENSORY NERVES

The function of the touch-, as well as of the warm-, and cold-spots is to initiate specific impulses that pass to the brain over paths that are anatomically separate as Cases 1 and 2 abundantly show. In the absence of the gross affective (hurt) element, as occurred in Cases 1 and 2, extreme stimulation of these spots never results in anything other than the corresponding specific sensation. Thus in Cases 1 and 2, in certain areas, ice, 70° C., and intense pressure, resulted in nothing more than the not unpleasant specific sensation of cold, warmth, and touch, respectively. It is obvious, therefore, that for all grades of stimulation each of the forms touch, warmth, and cold, is specific. It is further obvious that the ranges of stimulation for each of the forms constitutes a continuum. The "epicritic ranges" and "protopathic forms" of Rivers and Head (20) are, therefore, as the author has recently pointed out, (21) not only arbitrary and misleading but lacking in factual basis. All forms of sensibility with spotted distribution in the skin are fundamentally critical and specific with regard to the type of stimulus that is adequate in contrast with gross affective sensibility (hurt element) whose receptor mechanisms are practically spread ubiquitously over the periphery and capable of responding to different kinds of potentially noxious stimuli. Pain properly so-called is not, therefore, a transmutation of touch, warmth or cold as such, but the direct and immediate result of activation of the affective receptor mechanisms by tactual or thermic stimuli in which the potentially noxious element has attained threshold value.

TWO KINDS OF AFFECTIVE ELEMENT, THALAMIC AND CORTICAL

In the gamut the ranges toward the left are preponderately critical as compared with the ranges toward the right, the former (critico-affective forms) being mediated preponderately through the outer cortical mechanisms, the latter (affectivo-critical forms) preponderately through the inner cortical and thalamic mechanisms. It will be noted that pointedness, warmth, and cold, which are qualifying affective elements respectively in the higher cortico-affective forms (touch-point, touch-warm, and touch-cold) take on a relatively critical (inhibitory) function in the lower critico-affective forms

(point-hurt, warm-hurt, cold-hurt), a fact of fundamental significance in sensory-psychic integration. Two types of affective element are therefore distinguishable: (1) the gross affective (hurt) element mediated by paths that end in the thalamus and inner layers of the cerebral cortex, and (2) the higher affective element, *e.g.*, pointedness, warmth, and cold, in the higher critico-affective forms (touch-point, touch-warm, touch-cold) mediated by paths that end in the cerebral cortex preponderately in the outer layers. These higher critico-affective forms are to be regarded as psychic elaborates rather than simple primary sensory elements strictly so called. Each of the special senses carries an equivalent for the affective element of the higher critico-affective forms. As already stated, this is not a true fundamental sensory element but a psychic elaborate representing a transition from sensory affective element to affect properly so called.

DISPLACEMENT OF AFFECTIVE ELEMENT

Warmth and cold are closely related to each other as psychic alternates or opposites. This is a fact of significance in the mechanism of such phenomena as paradoxical heat and paradoxical cold. Thus during the progress of recovery after peripheral nerve injury, when sensibility for the hurt element of let us say 55° C. has recovered, that for the specific critical element still remaining in abeyance, the subject may call the stimulus (55° C.) cold. Here the affective element is actually transferred from its own specific critical associate (warmth) to its allied psychic alternate cold. Compare the paradoxical "heat" found in Case 3 on the sixth, ninth and tenth day. Psychologists are wont to explain the mechanism of this phenomenon by saying that heat can activate the specific mechanisms mediating the critical element cold. This is a fallacy if warmth and cold be regarded solely from the physiological side, *i.e.*, as pure elemental forms of sensation. If, however, warmth and cold be taken as psychic alternates (opposites) the mechanism of paradoxical cold (and heat) becomes intelligible as a psychic phenomenon in which the affective element entering consciousness without its natural critical associate (warmth) attaches itself to the closely related psychic alternate (cold). The dissociation of the affective element from its natural critical associate and its subsequent reappearance in consciousness, linked to a more or less remotely related critical associate, are facts of fundamental significance in sensory-psychic integration and furnish the key to the physiological mechanisms of many normal and disordered psychic states. Compare the phenomena of

displacement, suppression, conversion, etc. Without the aid of the dynamic affective element no critical element can enter consciousness. This seems to be a basic law of sensory-psychic integration. An affective element may, however, under certain circumstances, force its way into or remain temporarily in consciousness unattended by its natural critical associate. As such an event tends to psychic anarchy, an attempt to restore order is made by linking the unattached affective element to a fictitious, more or less unnatural critical associate. This temporary adjustment, consisting of "displacement of the affect," leads to all sorts of strange performances on the part of the subject in normal and abnormal conditions. It represents a defense reaction in the interests of internal (thought) orientation and ordered mentation. Under certain circumstances also an affect properly so called becomes transformed into an affective element of the lower or preponderately sensory type. This is the reverse of the process whereby the affective element of the affectivo-critical extreme of the gamut becomes transformed into the affective element of the higher critico-affective extreme of the gamut. Such degradation or downward displacement of the affective element from the higher psychic to the lower sensory or affectivo-critical level is a main factor in some of the more complex defense mechanisms. Compare the conversion phenomenon in which the affect, *e.g.*, the chagrin of a disagreeable experience, becomes in the hysterical subject transformed into the preponderately physical symptom pain.

THE JAMES-LANGE THEORY OF THE EMOTIONS AND THE REACTION ARC HYPOTHESIS

Pain is the psychic adjunct (equivalent) of activation of the afferent noci-ceptive neurones and not merely, as Sherrington (36) puts it, the psychical adjunct of the noci-ceptive reflex. Though necessarily a consequent of it in time, because of the distance the afferent impulses have to travel to reach the brain, pain is not causally dependent on the effector activities represented in the noci-ceptive reflex. It follows, therefore, that pain, and fear, its relative of near kin on the emotional side, are primarily and fundamentally dependent upon *direct sensory (affective) stimulation* and not merely upon secondary afferent impulses initiated by effector activities. In recent times too much emphasis has been laid by physiologists upon the rôle of muscle and gland effectors, to the exclusion of the direct effects of sensory stimulation, in the initiation of conscious phenomena. Undoubtedly thought processes and emotional states are generally, if not universally, associated with muscle and gland dis-

turbances (activation or inhibition) in some part or parts of the body, but this by no means proves that the muscle and gland disturbances are the primary, fundamental cause of the thought processes or emotions accompanying them. Psychologists, more especially those of ultra-scientific bent, have in recent years somewhat hastily accepted the erroneous conclusions of the physiologists in this matter. One result of this was the Lange, more commonly known as the James-Lange theory of the emotions which overemphasizes effector activation in the mechanism of the emotions giving this factor temporal and causal priority over all others. According to this theory as James (37) somewhat picturesquely puts it, "You do not run because you fear but you fear because you run; you do not weep because you are sorry but you are sorry because you weep." All of which strongly suggests putting the cart before the horse. Another result was the reaction-arc-hypothesis, or, as Dewey (35) prefers to call it, the organic circuit concept according to which as one modern psychologist puts it the muscle spindles (receptors) are "the beginnings of the thought arcs." This hypothesis is the near kin of the Lange theory of the emotions and is open to the same objection, viz., that it stresses inordinately the importance of the effector mechanisms in the initiation of conscious phenomena at the expense of the direct effects of sensory (affective) stimulation. The sensory repercussion initiated and perpetuated in the muscle-, gland-, and other receptors by effector activities undoubtedly plays a complementary rôle in extending and intensifying existing emotions but this is insignificant compared with the rôle played by direct sensory (affective) stimulation, somatic or splanchnic, in the normal mechanism of emotional excitation. The "emotive phenomena" attending the psychic or psycho-galvanic reflex as studied recently by Waller (19) and the contractions and other activities which the physiologist Mosso (34) and others observed in the urinary bladder and other viscera, animal and human, following so-called purely psychic stimulation have been overestimated by psychologists who did not apparently realize the significance of the relations, afferent and efferent, between the sympathetic (autonomic) and the cerebrospinal nervous system. The splanchnic sensory nerve supply is derived mainly, if not exclusively, from the affective system. Because of this fact the viscera undoubtedly play prominent rôles in initiating conscious sensations markedly affective in character. It may be admitted that effector activation in the viscera accompanies emotional states and is capable of adding to these or even of evoking in a secondary manner new emotional states but this is far from *saying*,

not to mention *proving scientifically*, that the sympathetic (autonomic, vegetative) nervous system plays the fundamental rôle in the mechanism of the emotions or, as Mosso (34) puts it, is the "seat of the emotions." Here it seems that physiologists and psychologists alike must be charged with a loose use of terms in so far as they confound the commonly accepted visceral effects of emotional states with the *status* of the person affected by the emotions. Such misapplication of terms too often masks the operation of the old fallacy of sliding or ambiguous middle term by means of which the most absurd conclusions seem to be rigorously deduced. In normal individuals the range of the emotions springing directly from activation, primary or secondary (effector activation, etc.), of the visceral receptor mechanisms is limited, being confined for the most part to the grosser emotions (fear, etc.) more immediately related to physical injury and pain. Physiologically it can be readily demonstrated that the visceral activities are powerfully affected by the emotions. The complementary rôle played by the viscera in the mechanism of the emotions has important clinical significance which as yet has not perhaps been adequately appreciated by practitioners of medicine; but this does not warrant the conclusion that the sympathetic or vegetative (autonomic) system is the "seat of the emotions." The emotions strictly so called are conditioned primarily and fundamentally by activation of the two great systems of afferent paths, viz., the affective and critical, functioning side by side in a coöperation of antagonism. One of these systems, the affective, terminating preponderately in the thalamus and inner layers of the cerebral cortex, plays the leading rôle on the physiological side in the mechanism of the coarser instinctive emotions (panic-fear, etc.). The other, the critical system, terminating preponderately in the outer layers of the cerebral cortex plays the predominant rôle on the physiological side in the mechanisms of the higher emotions. As neither of these systems ever functions wholly independently of the other, the co-operation of antagonism under which they function in the brain represents an interaction between correlating (association) mechanisms located at different levels, *e.g.*, between the mechanisms located preponderately in the outer cerebral cortex (critical system) and those located preponderately in the thalamus and inner cerebral cortex (affective system). This interaction, involving as it does the simultaneous activation of correlating mechanisms situated, to a greater or less extent, at all levels from the thalamus to the outer cortex, supplies the fundamental basis, on the physiological side, not only for the wide and varying range of the emotions but for the

phenomena of consciousness itself. It is a mistake, common amongst modern psychologists, to consider the emotional element (affect) as a thing apart from the critical or ideational elements. The fact is that every neural activation capable of attaining to, or remaining in, consciousness (awareness) must of necessity contain, in greater or less degree, actually or potentially, representatives of these two elements. In the coöperation of antagonism under which these elements function the affective element seems to be the fundamental determinant of consciousness.

SENSORY-PSYCHIC INTEGRATION

The deep critical system functions mainly as part of the sensory component in the postural changes incidental to spatial orientation and locomotion. In this capacity the deep critical system plays a fundamental rôle in the cognitive mechanism of protection. The superficial critical system on the other hand mediates the critical elements of ordinary skin sensibility (touch, warmth, cold, etc.) and of the highly specialized forms (vision, hearing, smell, taste, etc.) most of which are more immediately concerned in the physiological mechanisms of the higher psychic activities. On the physiological side the "psyche" is nothing more than a complex elaboration of the sensory mechanisms, the first step in such elaboration being inhibition of the affective by the critical mechanisms. With this established, cognitive as opposed to the more primitive instinctive modes of protection (flexion reflex, etc.), becomes possible. With these in operation ordered sensation ensues. This is the fundamental source of our ordinarily acquired knowledge and the immediate and essential prerequisite for the activation and development of the psychic faculties properly so called (intellect, memory, will, etc.).

TRANSITION OF AFFECTIVE ELEMENT TO AFFECT

The "psyche," therefore, in its physiological aspect represents a complex superstructure of mechanisms resting upon the lower sensory mechanisms as its foundation. Compare the transition of the gross affective (hurt) element at the affective end of the sensory gamut and mediated mainly by the thalamus, with the higher critico-affective element (warmth, cold or pointedness) at the critical end of the gamut and mediated mainly by mechanisms located in the outer layers of the cerebral cortex. The affective element in these higher critico-affective forms (warmth, cold, pointedness) is in reality a critical element, *i.e.*, an affective inhibitor when compared with the affective element in the affectivo-critical forms. It represents a

transformation in which the hurt element is actually absent although *potentially present* through fundamental and permanent functional association. The transformed affect, as previously pointed out, is more of a psychic elaborate than a pure sensory element as such. Although mediated by a special subdivision of the critical system, served by its own specific receptor mechanisms, nevertheless, in the intact individual, it does not enter consciousness without the coöperation of at least *potential appeal* to the affective system. With its varying shades of appreciable differences it represents a higher degree of psychic elaboration than that found in the affectivo-critical forms where the chief, and often the sole actual psychic adjunct is "it hurts." As a psychic elaborate warmth implies comparison at least of the concrete variety. At this level, however, the strictly psychic possibilities are extremely limited, being confined to concrete experiences and present, actual, sensory activation. This sensory-psychic level seems to bound strictly the psychic capacity of the lower animals. Physiologically it would seem but a step from the affective element of the higher critico-affective forms to affect properly so called. But here intervenes the "unbridgeable chasm" of the ancient psychologists apparently not so utterly unbridgeable, for on the physiological side the affective element of the higher critico-affective ranges undoubtedly merges into affect proper. On the physiological side, therefore, the mechanisms mediating our emotions form a continuum ranging from the comparatively simple affective system mediating the grosser emotions, panic, fear, etc., to the higher, complex, cerebral mechanisms mediating the subtle, more refined, critical "intellectual" emotions. The physiological mechanisms underlying psychic phenomena may with reason be regarded as elaborations of the two great divisions of the sensory nerves, viz., critical and affective—working in a coöperation of antagonism which supplies the physiological foundation for the dynamic complexities of normal and abnormal mental states.

PROTOPATHIC DISSOCIATION AND DISORDERED PSYCHIC STATES

As the "psyche" on the physiological side represents an elaboration of sensory mechanisms it is not surprising to find in psychic disorders a close analogy between the abnormally functioning affective system released from the inhibitory control of the critical mechanisms and the functioning of the corresponding *psychic* derivatives released from the control of their critical coöperatives. The condition of the affective neurones in protopathic dissociation

with their hyperelaboration and storage of neuro-potential, their raised initial threshold, and their tendency to react inordinately according to the "all or nothing" principle bears a close analogy to the phenomena of suppression, compulsions, and emotional outbursts. The other protopathic characteristics are also highly suggestive, the radiation and reference having their analogues in the phenomenon of *displacement*, and the persistence, poor localization and inability to name the stimulus, in obsessive thoughts and fears and in *internal (thought) disorientation*. Finally, the over-reaction which is the outstanding protopathic characteristic has its counterpart, of near kin, in compulsory states, emotional upheavals, and similar phenomena so common in psychic disorders. In the peripheral nerves where protopathic dissociation follows moderate degrees of trauma from such generally acting causes as toxemia, the mechanism consists mainly of injury and conduction block in the critical system with consequent release of the affective system from critical inhibitory control. Under these circumstances the hyperelaboration and storage of neuro-potential by the primary noci-ceptive neurones as well as the reversion of these latter to the "all or nothing" principle of functioning, represent a defense reaction intended to safeguard the individual against noxious contact with the environment. And as the "psyche" on the physiological side represents an elaboration of the sensory mechanisms, it follows that psychic phenomena such as are represented in suppression, upheavals, displacement, etc., are, at bottom, on the physiological side, nothing more than dispositions of neuronal activities with purposive mechanisms closely comparable to those found in protopathic dissociation.*

CONCLUSIONS

1. The sensory pathways in the spinal cord fall into two anatomically and functionally distinct divisions, the *critical* ("intellectual") and the *affective*, each being further subdivisible into a *superficial* and *deep* set of pathways giving in all four sets or systems, viz., (1) the *superficial critical*, (2) the *superficial affective*, (3) the *deep critical*, and (4) the *deep affective*.

2. Spinal cord lesions may cause, in the superficial or deep system, dissociation (1) of the *protopathic type* in which the critical

* Since this paper was written the author has read *Instinct and the Unconscious* by Rivers and is not surprised to find many points of coincidence between that observer's views and his own. For reasons stated in a previous contribution (21) the terms protopathic and epicritic as referring to forms of sensibility should be discarded and the attempt of Rivers to resurrect and introduce them into psychology is to be deprecated.

elements are suppressed or impaired, the gross affective (hurt) element being retained with protopathic characteristics; (2) of the *critical type* in which the critical elements are preserved, the affective (hurt) element being suppressed.

3. Each subdivision of the critical system exercises inhibitory control, total and fractional, over the corresponding subdivision, total and fractional, of the affective system; this represents the first step towards ordered sensation and psychic integration.

4. In sensory integration the optic thalamus is the lowest level at which the critical impinges upon the affective system; at this level the critical system directly exerts its main fundamental inhibitory control upon the affective system.

5. "Tract irritation" is insufficient to explain the mechanism of obscure pains of spinal origin.

6. Critical dissociation attends cord lesions extending from the central canal outwards thereby abolishing conduction primarily in the affective system.

7. In studying the mechanism of the nerve impulse, more especially in relation to the phenomena of consciousness, or of the emotions, the status of the neurone bodies in the dorsal root ganglia is a matter of prime importance.

8. Protopathic dissociation attends lesions that compress the cord from without inwards; such lesions abolish, partially or completely, conduction in the critical system thereby releasing the affective system from the control normally exerted upon it by the critical system.

9. A further factor in the mechanism of protopathic dissociation is hypermetabolism and hyperfunctioning, more especially in the bodies of the primary affective neurones, representing the attempt on the part of these latter to overcome conduction block in the functionally related long tracts (lateral spinal lemniscus, ventral spinal lemniscus, etc.).

10. Each subdivision of the critical system functions totally and fractionally in a coöperation of antagonism with the corresponding subdivision of the affective system, each contributing its elemental quota to the synthetic conscious resultant.

11. In actual experience the gamut of our sensations runs:

<i>Higher</i>	<i>Lower</i>	
<i>Critico-Affective</i>	<i>Critico-Affective</i>	<i>Afectivo-Critical</i>
Touch-point	Point-hurt	Hurt-point
Touch-warm	Warm-hurt	Hurt-warm
Touch-cold	Cold-hurt	Hurt-cold

12. All our general sensations are resultants of the gross affective or hurt element, coöperating, actually or potentially, with one or other of the fundamental critical elements.

13. Each of the higher critico-affective or elementary forms (touch, warmth and cold), as well as the fundamental affective element (hurt), is mediated by its own set or system of pathways served by its own set of receptor mechanisms.

14. The receptor mechanisms of each of the higher critico-affective or elementary sensory forms (touch, warmth and cold) are specific for their own form, whereas the receptor mechanisms of the affective system are communal in function responding to tactile and thermal stimulation when the potentially noxious element reaches threshold value.

15. The doctrine of functional specificity (specific energies) holds for the higher critico-affective forms (touch, warmth and cold) both as regards receptors and pathways, whereas, in the affective system, it holds for the pathways but not for the receptor mechanisms although it is strictly specific for the noxious element.

16. Pain is not a transformed (intensified) tactual or thermal sensation but a pure elemental form resulting from direct activation of the affective mechanisms by noxious stimulation.

17. Normally no critical element tends to enter, or remain in, consciousness without modification by an affective element; and conversely no affective element tends to enter or remain in consciousness without modification by its critical associate.

18. The higher critico-affective forms (pointedness, warmth and cold) although they represent fundamental sensory forms of actual experience are psychic elaborates rather than pure sensory elements.

19. The transition whereby the affective element (hurt) in the affectivo-critical forms becomes represented by a non-nocuous sensory affective element in the higher critico-affective forms (pointedness, warmth, cold) represents an important step, on the physiological side, towards the elaboration of affect properly so called.

20. As the "psyche" on the physiological side represents an elaboration of the sensory mechanisms it is not surprising to find many similarities in the mechanisms underlying sensory dissociation and those underlying sensory-psychic integration.

21. On the physiological side the phenomena of normal and abnormal psychic states, representing at bottom dispositions of neuronal activities, have their close counterparts in the dissociation

phenomena consequent on nerve injury or functional block mainly in the critical sensory pathways.

22. Effector activation, and the secondary afferent impulses arising therefrom, are unduly emphasized in the James-Lange theory of the emotions and in the "reaction-arc" or "organic circuit" hypothesis at the expense of direct sensory (affective) stimulation which is the main factor, on the physiological side, in the initiation of consciousness and the emotions.

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244 West 101st Street.

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SOCIETY PROCEEDINGS

NEW YORK NEUROLOGICAL SOCIETY

THE FOUR HUNDRED AND ELEVENTH REGULAR MEETING, MARCH 4,
1924, PRESIDENT DR. E. G. ZABRISKIE PRESIDING

I. TWO UNUSUAL CASES OF MYOPATHY

DRS. IRVING H. PARDEE and I. MARGARETTEN

Dr. Margaretten reported Case I. L. G., Hebrew, age seven, had been under the observation of the Neurological Institute for four years. The complaint was that he was disobedient, stubborn, inattentive, destructive and masturbated. He was generally weak, unable to walk up or down stairs, to hold up his arms or to grasp firmly. He had difficulty in swallowing and chewing; pain in the back of the neck; headache; sleeplessness and constipation; was a mouth breather. These symptoms had gradual development from infancy, the mental symptoms predominating. The patient had an instrumental delivery, was weaned and had first teeth at six months; all teeth at eighteen months; walked at fourteen months; had colic as a baby; also fell against the piano, striking his head, with no sequelæ.

In October, 1919, the mother stated that the child was incorrigible at home, kicked, screamed, bit visitors and was destructive; that he got tired after a short walk and would ask to be picked up. About January or February, 1923, she said there was an abrupt change in his condition. His mental state subsided somewhat with a general weakness. He was strangled by a rope around his neck and a few days afterward the weakness in the extremities became definitely apparent. The lower extremities were first affected and very recently the upper extremities and eyes and chewing muscles became involved. The mother stated that the muscular activities were much weaker in the afternoon. About January the mother noticed a lump in his neck which had progressively grown worse. He saw double at one time, but not lately. Speech and sphincters were not affected, although some of the relatives state that he never articulated well. For six weeks he has complained of recurrent headaches which lasted a few days and then disappeared. While in the hospital these symptoms grew progressively worse. He was much stronger in the morning. There was less drooping of the eyelids. He fed himself more readily and swallowed, but choked even with milk. He was restless during the day as well as during the night, used profane language and was very untidy and disobedient. He left the hospital unimproved. After leaving, as he was gulping some tea one morning he coughed and choked until he expired.

Physical examination, April 28, 1923: eyelids droop, mouth open, teeth irregular, walks on toes; acts the clown, mischievous; coöperates but is inattentive and restless; has a weak hand grip. He is able to squat and rise, but when lying down gets up weakly and awkwardly. Reflexes, sensation, cranial nerves, associated, automatic and alternating movements and coördination were normal. Cardiovascular apparatus and viscera were normal.

A note on January 3, 1924, states that he is a well-nourished and well-developed boy of seven, sitting on the bed with head drooping forward; expressionless, flat, sallow, adenoid facies, ptosed eyelids.

When standing he assumes an attitude of dystrophy, *i.e.*, genu recurvatum and a lordosis. He has a female outline of the chest and pelvis and a smooth, velvety, puffy skin, partially descended testicles, faint temporal eyebrows, high arched palate and spaced ragged teeth, small lateral incisors and a large, thick tongue. He is able to stand or walk but soon tires and sinks to the floor. He is unable to sit up and gets fatigued after a few attempts. He cannot hold head erect, it falls forward or backward. There is marked hypotonia and asthenia in all muscles. There are no abnormal movements. Sensation is normal. All reflexes are hyperactive and become fatigued after repeated testing. There is a static Babinski attitude of the toes. Cranial nerves, with the exception of the motor, are normal. There is a bilateral ptosis of the upper lids. There are nystagmoid twitches in all extraocular movements. Jaw, facial, tongue, pharyngeal and laryngeal muscles are all weak. There is no atrophy or hypertrophy anywhere. Faradic stimulation shows the myasthenic reaction. There is an enlarged thyroid with a nodule on the left lobe.

In the laboratory findings there is a relative lymphocytosis 52 per cent, a secondary anemia of 70 per cent. The Wassermann is negative in the blood and spinal fluid. Urine, highly acid with a high specific gravity. An X-ray of the chest does not show an enlarged thymus. X-ray of the skull shows a small sella, roughened outer edge and a heavy dorsum. Intelligence quotient in three tests was always 100 per cent. In view of a gradually progressive condition, a pathological thyroid ancestry, and symptoms of a pituitary, thyroid, thymus, adrenal and gonadal decrease, we consider the myopathy the result of an abiotrophic state. The mental and myasthenic symptoms may be in favor of encephalitis but there were no remissions such as is common in encephalitis.

Dr. Pardee presented Case II. Girl, Jewish parentage, aged thirteen years, arms and legs have become progressively weaker; ptosis of eyelids is present. The reflexes are normal. The girl is fairly intelligent, rather apathetic. She was under treatment in the Beth Israel Hospital where she was supposed to have congenital syphilis on account of a positive blood Wassermann. This is the only time it was found. The Wassermann in the parents is negative. The girl presents signs of an endocrine imbalance.

Discussion: Dr. H. A. Riley said: Is there any fatigability of the jaw jerks?

Dr. I. H. Pardee said: No, there is not. We were not able to demonstrate fatigability of the muscles but she says there is variation of the muscles. I have seen more ptosis, especially under emotional stress, as in being in this room. There has been no diplopia at any time. The abdominal reflexes are present.

Dr. E. G. Zabriskie said: In regard to Case II, it seems chiefly remarkable for variation in fatigability of the musculature.

Dr. Grimberg said: I would like to ask if there were any other reports of a positive Wassermann except for that first one at the hospital. You say the Wassermann reports on the parents were negative.

Dr. Abrahamson said: The case shown by Dr. Margaretten impressed me as a myasthenic type of a chronic lethargic encephalitis; the bulbar death, the mental changes, the variability of the myasthenic phenomena and the tendon reflexes. Some type of endocrinopathy was present, but I do not regard it as the cause of the manifestations.

Dr. Pardee (closing) said: In answer to Dr. Grimberg, the patient had a positive Wassermann test in the Beth Israel Hospital. Treatment was instituted there as the case was considered luetic. The presence of positive serum and positive Wassermann is suggestive of luetic infection, but I do not think we are in a position to say that she had lues; if she had, it was only another condition associated with the present disturbance. I am glad to get Dr. Abrahamson's point of view because I have seen cases of his type following encephalitis. I am glad he mentioned this as we had thought of it, but we were not able to make such a definite statement as Dr. Abrahamson. From the standpoint of endocrine imbalance, she does fit into the type of status thymicolymphaticus. This does accompany myasthenia gravis. Sixty per cent of these patients show persistent thymus at autopsy. This, combined with the very low blood pressure, fits her into that group very definitely. I am interested to watch her progress. The next two or three years will settle the question of diagnosis.

II. NEUROEPITHELIOMA OF THE SPINAL CORD, WITH CLINICAL AND PATHOLOGICAL NOTES

(Lantern Illustrations)

DR. JOHN A. HARTWELL (by invitation) and DR. LEWIS D. STEVENSON

The tumor used as the text of this discussion was entirely extra-medullary, originating beneath the pia arachnoid, the latter membrane passing over it and binding it closely to the cord. It was excessively malignant in type. A gliosis appeared, but seemed to be of later date than the tumor.

The patient in whom the tumor developed was a woman of forty. She had sustained an injury to the cervicodorsal spine, two years previous to onset of symptoms. The outstanding symptom was

subjective pain in the distribution of the brachial plexus and the second and third dorsal nerves, at first recurrent, but not severe. Later it was very severe with no relief. She was first observed in March, 1922; at that time there was tenderness over the second and third dorsal spinous processes and an area of hyperesthesia in the distribution of these nerves. There was some interosseal atrophy in the left hand. There were no alterations in the reflexes, no other paresthesias, and no motor changes. The subjective pain was excruciating, without remission. On May 26th there was sympathetic involvement on the left side, shown by false ptosis and contracted pupil.

The diagnosis favored by consultants was cervicodorsal radiculitis with pachymeningitis, possibly due to infection from a tooth root. The question of neoplasm was raised but evidence therefor was not deemed conclusive. Pain continued through the summer; in September, the right scapular region became painful; in October there there first appeared evidence of cord involvement. There was momentary weakness in the left lower extremity and paresthesia in the right thigh. In ten days the picture had passed through a Brown Séquard syndrome to an almost complete transverse lesion of the cord at the first and second dorsal segments.

Operation: Laminectomy, discovering the tumor lying beneath the arachnoid, with maximal pressure at this region, and spreading upward and downward for a total distance of 10 c.m., i.e., from Cervical V to Dorsal III. There was also a very small tumor found in front of the cord in a pit-like depression in the dura.

Pathological Report by Dr. Elser: New growth, soft, pliable, tongue-like piece of tissue, 4.5 c.m. long, and 1.25 c.m. wide, 4 mm. thick, tapering off towards the ends. The tissue resembled splenic tissue in appearance. The outer surface was smooth and glistening, the under surface coarsely granular. The growth appeared to be composed of congeries of blood vessels, leading to a tentative diagnosis of endothelioma. The smaller tumor was isolated, and to the eye, not connected with the main growth. It was partly blood clot, partly tumor tissue.

Postoperative Course: The wound healed by primary intention, and there was definite improvement in all ways for about four weeks. Thereafter there was rapid retrogression, with varied cord manifestations, until death, seven weeks post operative, from complete transverse myelitis.

At autopsy, a recurrence of the tumor measuring 4 cm. and 5 mm. thick was found, and strikingly resembling the original tumor. There was a compression myelitis of the cord. There was considerable straw colored fluid collected below the site of the operative field, otherwise the cord below appeared normal. Bacteriological examination of this fluid was negative.

A detailed pathological examination of the cord and tumor was made by Dr. Stevenson. The pathological process coordinates with the clinical course. While there were intervals after the trauma when

symptoms were not present, subsequent developments showed that the lesion coincided closely with the site of injury. It appears that nerve tissue may be more prone to start tumor formation than other tissue.

The injury (fall from a horse) occurred in November, 1919, and was followed by only a transitory inconvenience a few days later, apparently due to muscular and joint injury, rather than any damage to nerve elements. Twenty months later, the first pain, evidently due to nerve change, appeared, was present two months, disappeared for five months, and after that appeared with remissions, but no complete relief, until at nine months, evidence of cord compression appeared. A period of some months after the injury elapsed before the cells lost their normal restraints and took on malignant characteristics. On cord section Dr. Stevenson found evidence of central gliosis, but as no symptoms referable to this were evident, we are forced to conclude that the gliosis took place after the formation of the neuroepithelioma. (This is contrary to the conclusions of Schlaff.)

The question whether earlier operation would have saved the patient is pertinent. She was under observation for months but showed no symptom which was incompatible with an inflammatory lesion of the nerve roots. It was felt wise to defer operation until some clinical evidence of implication of the cord was obtained. A true diagnosis cannot be made until medullary symptoms appear. At no time would removal have been accomplished with less trauma or greater certainty of removing all potential tumor elements. The future must determine the final value of radiotherapy, but our present knowledge holds out at least a hope that these tumors are susceptible of destruction by radioactive agents.

Discussion: Dr. Foster Kennedy said: I think it is in order to mention how beautifully this case has been presented, and the excellent and thorough work done by Dr. Hartwell and Dr. Stevenson. The account of the beginning of the patient's clinical symptoms, to the high power photographs which we saw,—there is nothing wanting in the examination and in the presentation of the facts. It has been done with an infinite amount of care. I know of the work expended upon the case by Drs. Hartwell and Stevenson, and the result has justified the pains they took. The facts that Dr. Hartwell put forward are indisputable and when I was confronted on October 11, with clear evidence that we were dealing, not with hypertrophic cervical pachymeningitis, to which view I had, with a certain amount of obstinacy adhered, I felt that I had made a blunder. Of course, now, in the light of our after knowledge each symptom and sign appears definite, and one cannot go back to the standpoint one had before knowledge was complete. When the facts were once clear, we operated within a week. I am sure now I should have advised Dr. Hartwell to operate when ptosis and small pupil occurred. Of course sympathetic paralysis might have resulted from hypertrophic pachymeningitis influencing the spinal roots. Had we operated we

should have found a very small tumor playing the same rôle as inflammatory exudate, but I believe in any case the final result would have been the same.

Dr. E. G. Zabriskie said: I would like to ask Dr. Hartwell, from the point of view of his experience, to take up the question of what possible influence an earlier operation would have had on the outcome of the case, whether it would have saved the patient or not, and avoided the final event. What, also, were the findings in the spinal fluid.

Dr. John A. Hartwell said in closing: In answer to the first question, I do not think earlier operation would have saved the patient. In view of the fact that the very small tumor removed from the pit in the anterior dura and recurred in seven weeks to a large size, and the fact that at autopsy the whole neighboring dura and intervertebral foramina were infiltrated with tumor, and the exceptional malignant character of the tumor, as pointed out by Dr. Stevenson, I do not feel that operation would have been more successful if performed earlier. As to the information obtained by an earlier spinal puncture I can only express the belief that none would have been forthcoming until pressure symptoms developed.

I would like to relieve Dr. Kennedy's mind as to why he was not able to arrive at a definite conclusion earlier. The patient was seen by several men who expressed the opinion that there might be a neoplasm, but no one of us could arrive at that conclusion. In view of this doubt we hesitated to do an exploratory operation in the cervicodorsal region, which, as you will understand, is a procedure of considerable danger. As to what could have been accomplished by radiotherapy, this is a matter largely of conjecture. Undoubtedly, this type of tumor would be influenced by radioactive measures, but as to whether it could be cured is quite another matter. After careful consideration it was decided that in view of the doubtful diagnosis, operation offered more chance of relief than radiotherapy. We desire to express our thanks to Drs. Ewing and Elser for their great help in our study of this case.

III. ON ASTEREOGNOSIS

DR. FOSTER KENNEDY

(Author's Abstract)

Astereognosis (impaired ability to find the dimensional quality of the object) should not be confined to the cortex alone; it may be thalamic, peripheral, spinal, or cerebral. I believe that this lack of function should be localized in clinical reports and that in examinations, one should be very careful to make sure what the subsidiary effects are in these cases. Astereognosis should be classified according to the physiological or anatomical level of the sensory defect, and remarks should be qualified with the anectant sensory losses when describing the condition.

Discussion: Dr. Henry A. Riley said: Dr. Kennedy has given us a very interesting presentation. I can recall Cushing's views ex-

pressed at the meeting of the American Neurological Association. To me the term astereognosis means the inability to recognize objects without the use of the eyes and in the presence of unaffected primary sensibility, which places it above the lower level and in the psychic areas of the cortex. As Dr. Kennedy pointed out, the faculty to recognize objects may be distorted by the abrogation of any of the constituent parts of sensation. I believe the term stereognosis gains greatly in value by limiting its application to the pure critical activity of the recognition of objects by the sense of feeling without the use of the eyes in the presence of unimpaired primary sense qualities, and that its loss, namely astereognosis, should be likewise restricted in its application.

Dr. Abrahamson said: The phenomenon called astereognosis must first of all be differentiated from asymbolia; second, there are two reflex arcs, an upper and a lower one, and in speaking of astereognosis, these have to be constantly kept in mind, especially where we wish to use it as a localizing sign.

Tonus orientation, being related to the cerebellum, it seems to me that occasionally where this sensation is severely affected, the recognition of objects by this sensation would suffer also. This did occur in estimating the sensation of weight, as the butchers do, by balancing the object in the air and estimating the weight by the amount of effort necessary to overcome gravity. The cerebellum is the reservoir of sensory impressions from the muscles, tendons, joints, etc., and its part in the genesis of astereognosis has to be considered.

Dr. Wechsler said: Astereognosis is a specific psychological concept. Clinically it consists of inability to recognize objects and, as Head has shown, of loss of the sense of position and discrimination. All other forms of sensation are intact. The seat of the lesion is either cortical or subcortical (post central) or limited to a small area of the thalamus. Now, if we are to use astereognosis generically and include under it any loss of ability to recognize objects, irrespective of the accompanying sensory disturbance, we shall simply discard a term which has a very definite clinical, anatomical and psychological value. Obviously one cannot recognize an object if one has no sense of touch or pain and temperature, but the failure is not due to any conceptual defect. And just as we limit the term aphasia to speech disturbance in the presence of intact peripheral speech apparatus, both on the receptive and the expressive side, so should we do with astereognosis. Unless, therefore, one denies a higher psychological concept to the term or merely wants to use it generically, and then qualify it so as to associate the other specific sensory losses in order to localize the seat of the lesion, it seems that the more restricted use of the term is to be preferred.

Dr. E. D. Friedman said: Dr. Kennedy has given us a lucid explanation of this phenomenon. Strümpell has recently published an article on this subject. According to Wernicke the local area in the brain is in the lower arc alone. He calls astereognosis the inability

to recognize objects placed in the hand. According to Strümpell there is no astereognosis without disturbance of the posterior column. Occasionally from a focal lesion in the brain postural sense can be lost. The question of aphasia is similar, whether we indicate a local area in the brain which leads to aphasia, or whether other parts are implicated and aphasia results. Astereognosis is not strictly a cortical phenomenon, but can arise from other portions of the brain. Polyneuritis is a columnar involvement and in combined sclerosis there is loss of power with typical athetosis. Astereognosis can occur in lesions of the thalamus. Dejerine and Lucie do not limit astereognosis to lesions of the cortex.

Dr. Foster Kennedy (closing) said: In reply to Dr. Wechsler, it would seem as if he would limit the word astereognosis to purely cortical conditions. This is what Cushing did. He defined it as cortical and he had in mind a purely diagrammatical idea, that astereognosis was a cortical phenomenon and indicated the post Rolandic area. Dr. Wechsler says astereognosis is a cortical function. I think this concept would increase error in this question.

I believe these patients are always sensorially abnormal. You may have to move the finger in a wider angle to get the patient to recognize the object, or to put greater pressure; in other words, to give the patient more information in one way or another to find out whether he is kinesthetically accurate or not. You will find inability to recognize form only when other sensation defects exist.

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY FEBRUARY 21, 1924

DR. C. MACFIE CAMPBELL IN THE CHAIR

AN UNUSUAL CASE OF PISTOL-BULLET WOUND DR. J. W. COURTNEY

The subject of this communication was seen by me to-day at the Cambridge Municipal Hospital. He is a man in the late forties. As he was leaving a poolroom in Cambridgeport one evening last December, a foreign-looking individual armed with a revolver sprang toward him with the exclamation: "I've got you at last!" and at the same moment fired. There were powder stains on the victim's face. The bullet carried away the two upper median incisors, ploughed a furrow along the upper surface of the tongue and clipped off the uvula. An x-ray taken at the hospital shortly afterward showed the bullet, which was of heavy caliber, in contact with the anterior surface of the body of the second cervical vertebra. There were no cord symptoms of any kind or degree; in fact, surgical intervention was in nowise indicated.

Some days later an abscess began to point along the outer border of the upper third of the right trapezius. This was later evacuated by Dr. H. H. Germain who at the same time tried unsuccessfully to

get the bullet. An x-ray taken after this intervention disclosed the fact that the bullet had disappeared. It showed also some evidence of structural damage to the body of the axis.

The proximity of the original site of the bullet to the esophagus suggested that the missile had dropped into the stomach. As a matter of fact, an x-ray located it in the cecum. The stools were watched, but the bullet was not recovered. Nevertheless, a further x-ray showed that it had been eliminated.

The patient was discharged directly his operative wound healed. He has recently returned to the hospital, with increasing stiffness of the neck and severe pain which radiates along the course of the occipitalis major on either side. Neurologic examination is negative except that the palatal reflex is abolished. The speech nasal and deglutition painful. There is little or no febrile reaction.

My feeling is that we are dealing with a traumatic osteomyelitis of the axis, but a very recent x-ray provides no conclusive evidence on this score.

[Ultimate Outcome: The case ran along without notable change for a fortnight or longer after I saw it. The patient then went home, against advice, and died. The autopsy disclosed a purulent necrosis of the entire axis and a partial destruction of the third cervical vertebra. There was in addition a generalized purulent meningo-encephalitis.]

PITUITARY DISEASE

A symposium on pituitary disease was presented by members of the staff of the Peter Bent Brigham Hospital. Dr. Harvey Cushing's regrets for his inability to attend the meeting were read.

I. Presentation of cases by DR. GILBERT HORRAX:

Attention was called to the three classes of patients suffering from pituitary disease showing evidence of neighborhood or general pressure symptoms requiring surgical measures for relief.

Group A. Acromegaly

The history and clinical course of an acromegalic patient operated upon three days previously by Dr. Cushing was given by Dr. Tracy Putnam. This patient showed the bony and sellar changes typical of the condition. He complained chiefly of extreme weakness and lassitude. On two occasions he had had a transient temporary hemianopia of the left eye. He was given two therapeutic x-ray treatments with marked improvement in his symptoms. He was then sent home, and received at another clinic a third x-ray treatment. After this treatment there was almost immediately a great decline in vision, and he returned here for observation. When no improvement occurred after two weeks he was operated upon by Dr. Cushing by the transphenoidal route and a portion of the soft hypophyseal struma removed. Microscopic examination of this material showed almost complete necrosis. There was rapid return of vision following this procedure.

Patients illustrating the clinical picture of the next two groups were demonstrated by Dr. G. Horrax.

Group B. Dyspituitarism with Pituitary Adenoma and Large Sella

1. M. S., Surgical No. 20668, a woman of forty-five who entered the hospital complaining of failing vision. Her family and past history were irrelevant. Three years ago she began to notice a gradual loss of vision in the left eye, which progressed until one year ago when it was noted that vision on the temporal side of the left eye was entirely gone. At this time vision in the right eye began to fail, and continued to a total loss of the temporal field on this side also. She had not menstruated for three years.

Physical examination showed the presence of bilateral primary optic atrophy and a typical complete bitemporal hemianopsia. The x-ray showed a deep, irregular sella, with marked erosion of the posterior clinoids. The basal metabolic rate was 19 per cent below normal.

A transphenoidal operation with evacuation of a considerable amount of soft pituitary adenoma, was performed by Dr. Cushing on February 18. The patient showed an immediate improvement in vision after coming out of her anesthetic with demonstrable widening out of the right temporal field.

This case is typical in all respects of the group of patients presenting indications for a transphenoidal operation rather than an operation from above, the choice being made on the basis of a generally enlarged sella, without evidence of calcification either within or above it. In this connection, attention was called to another case in the hospital showing the same clinical picture as the patient just demonstrated. In the patient referred to, a previous intranasal operation had been performed on the basis that her symptoms were caused by some local condition. This previous operation, with removal of the septum, made it impossible to carry through the transphenoidal operation, an attempt at which had to be abandoned.

2. E. N. G., Surgical No. 20755, a young man twenty-five years of age, was referred to the hospital by Dr. G. E. Dailey, Gloversville, N. Y. His chief complaint was loss of vision in the right eye. Except for a tendency to epistaxis during the past six years, his past history was negative. His present illness began two years ago with frontal headaches and gradual failure of vision in the right eye. He noted from the outset that he could not see objects on the outer (temporal) side of the right eye. This failure of the right eye progressed steadily up to the present time. In July, 1923, a right turbinectomy was performed for relief of vision without effect. During the past three years he gained thirty pounds in weight.

Physical examination showed a moderately obese individual with soft, smooth skin, the hair and fat distribution over the body being distinctly of the feminine type. His beard was scanty. There was a well marked primary atrophy of the right optic disk, and a slight pallor of the left disk also. Visual fields showed the retention of only the upper nasal quadrant on the right, with a normal field for the

left eye. X-ray of the skull showed a generally enlarged and deep sella turcica, with extremely thin posterior clinoids.

This patient illustrated an outstanding hypopituitarism of the syndrome adiposo-genitalis with a large sella containing undoubtedly a soft adenoma, or so-called chromophobe struma. He was awaiting operation by the transphenoidal route and was believed to be an ideal case for this procedure.

Group C. Dyspituitarism with Suprasellar (Cranio-Pharyngeal Pouch) Cyst

1. T. H. C., Surgical No. 20737, was a child of five, referred to this clinic by Dr. L. C. Heridger of Bridgeport, Conn., because of headache, vomiting and blindness. The child's early development was apparently normal. Diminution of vision was first definitely noted when he was two years old, *i.e.*, three years ago, and at the same time it was seen that his eyes were becoming "crossed." Diminution of sight progressed to complete blindness by October, 1921, although he was still in good health and active. Fourteen months ago the child had some vomiting spells and drowsiness during the course of one week, and then was again active and bright until seven months ago. From this time until the present he had continued headaches and vomiting, marked loss of appetite, emaciation, drowsiness and general weakness.

Physical examination showed an emaciated and extremely lethargic child who could not be aroused by any ordinary stimuli. There was conjugate deviation of both eyes to the right with continuous nystagmus in this direction. Both fundi showed the presence of complete primary atrophy, upon which certain pressure changes had been superimposed. Stiffness of the neck was marked and Kernig's sign was positive. X-ray of the skull showed marked convolutional atrophy and a large, ballooned-out sella with thinned posterior clinoids. Within the sella there were multiple, small dense shadows of calcification, which were believed to be undoubtedly in a suprasellar cyst.

On February 19 the cyst was tapped by Dr. Cushing through a single burr opening in the skull. Following this procedure the child roused up, responded and took nourishment. Further taps of the cyst were contemplated with a view to improve the patient's general condition sufficiently so that a frontal operation could be performed, and the cyst radically removed.

Obviously in cysts above the sella, the only approach which offers any possibility of removal is by an intracranial operation. The frontal lobe can be retracted and the tumor exposed to some degree. In certain instances a complete enucleation of the cyst with its contained calcifications can be accomplished.

The following case was presented illustrating the same type of condition in an adult. This patient was in the early stages of pressure with headaches and choked disks. No specific glandular manifestations were present.

2. S. J. C., Surgical No. 20726, an Italian laborer of thirty, was referred by Dr. J. W. Courtney. His family and past history were negative. The present illness began one year ago with frontal headaches—supraorbital—which continued to the present time. There had also been tinnitus in both ears, and during the past six months an increasing unsteadiness in gait. He complained at times of a burning sensation on the left side of the face.

Physical examination showed slight bilateral exophthalmos; early choked disks, more marked on the right, and a relative hypesthesia of the left cheek. X-ray of the skull showed in addition to marked convolutional atrophy, a deepened, irregular and eroded sella, with some probable areas of calcification just above it. The basal metabolic rate was 14 per cent normal.

This patient was awaiting operation, and again illustrated the type of condition best dealt with by a frontal intracranial procedure.

II. A Critical Analysis of the Anamnesis in Seventy-two Cases of Acromegaly, by DR. LEO M. DAVIDOFF:

From the large group of cases with acromegaly in Dr. Cushing's records, seventy-two were selected for analysis because these were admitted to the surgical wards of the Peter Bent Brigham Hospital and were all subjected to a uniformly thorough investigation, established as a routine procedure at this clinic for the study of pituitary disorders. The physical characteristics of this disease are so familiar to any one who has ever seen even a single typical case, that this study confined itself largely to the anamnesis, which up to this time has held a secondary place of importance in the literature.

In spite of the exaggerated number seen at this clinic, the acromegals comprise only 0.2 per cent of all patients admitted to the wards. Yet granting its rarity, it seemed not only justifiable, but necessary, to continue the intensive study of acromegaly. For undoubtedly, any light that may be thrown upon a condition which involves so much of the personality of its victims, will help to dispel the mystery in which the entire group of deeply constitutional diseases is involved.

This analysis of a series of cases subjected to a uniformly thorough investigation in the wards of the hospital will subsequently be reported in full.

III. DR. PERCIVAL BAILEY presented the results of a histologic analysis of a series of forty-two cases of acromegaly from which material had been removed at operation. This material, consisting of fragments of the hypophysis, had been fixed immediately, and so was suitable for a microscopic study.

The material had been fixed in three different fixatives—Bensley's, Zenker's, and 10 per cent formalin, with the exception of the more recent ones, which had been fixed in Regaud's fluid.

Dr. Bailey insisted upon the necessity of investigating pathological alterations of the structure of an organ by means of the same methods used for the demonstration of its normal structure, as well as by the use of methods for displaying pathological substances.

An analysis was briefly given of staining methods as applied to the pituitary gland, the essentials of which were the necessity of avoiding acids and sublimate mixtures as fixatives, and of employing specific dyes for the demonstration of the granules. It was concluded that an optimum procedure consisted in fixing the material in Regaud's fluid and staining by means of ethylviolet-orange G for the alpha-granules and with acid-violet for the beta-granules. Double staining of the two granules in the same preparation might be done by the use either of Bailey's acid fuchsine-acid violet combination, or safranin acid-violet combination of Maurer.

From a study in this manner of all the material available, and a comparison of the results with the Zenker- and Bensley-fixed material, it was concluded that the hypophysis of acromegalics shows definite histological characteristics by means of which it may be identified. They will be described in detail in a paper soon to be published.

CURRENT LITERATURE

II. SENSORI-MOTOR NEUROLOGY.

5. CEREBELLUM, THALAMUS, BASAL GANGLIA.

Rombach, K. A. FRIEDREICH'S ATAXY. [*Nederlandsch Tijdschr. voor Geneeskunde*, 1921, Jan. 29, 504, 4 figs.]

Rombach's case presents some rather unusual symptoms. A man of thirty came for life insurance, showing an oscillating gait. At eleven he had an abscess in the neck which was opened. At thirteen he was "tired in the legs," at seventeen "rheumatic in the legs," and he had occasional vertigo. Good personal and family history, a maternal uncle diabetes, nothing like Friedreich's ataxy in family; paternal grandparents were first cousins. No stigmata of degeneration. Face pale, aspect suggests slight psychical inferiority, good intelligence. Slight scoliosis in dorsal region, marked lumbar lordosis, sacral kyphosis, occasionally slight nystagmus, definite on ocular movements. Slight articulatory disturbance. words are drawn out, often indistinct (drawling speech). Sometimes slight urinary incontinence. Wassermann negative. For two years salivation, formerly often had hyperidrosis, now seldom. Gait of cerebellar ataxic type, tendency to propulsion, patient says he often falls. Rombergism. When sitting, there is a very slight ataxic movement of the head. Arms not ataxic, no choreiform movements. Great hypotonia of joints. Writing natural, but if kept up for long becomes a scrawl. Absent ankle-jerks, feeble knee-jerks. Bilateral Babinski sign. Bilateral pescavus. Cremasteric reflexes not obtained, abdominals minus. No muscular atrophies, no vasomotor affection. Rombach discusses the various opinions held about Friedreich's disease. Changes occur in the dorsal roots and columns, the cerebellar tracts, and the lateral pyramidal tracts, and the cells of Clarke's column are degenerated; also changes occur in the ventral columns. Charcot and his school regarded the disease as a special form of disseminated sclerosis. Hammond favored its cerebellar origin, a combined sclerosis of the lateral and the dorsal columns could not explain the clinical picture, seeing that sensory changes are absent, the pure ataxia points to the cerebellum. Against this view, Jelgersma found that in many cases even microscopical examination failed to show any cerebellar changes. He leans towards Stcherbak's view that it is a disease of the peripheral connections of the cerebellum with the reflex arc. In the dorsal columns run two kinds of systems, the one goes over the dorsal column nuclei near the inter-olivary layer, and

reaches the cerebrum; in it the deep sensibility sense should be localized. The other system runs directly to the cerebellum. A lesion of this system gives ataxia without sensory disturbances. Therefore Friedreich's ataxy should depend on an affection of this system. Necropsies show the nervous system to be smaller than normal (sclerosis and atrophy); there is also chronic meningitis in the region of the dorsal columns. Jelgersma holds that the primary change is in the parenchyma, and this causes a secondary neurological proliferation. Recently Brouwer has made the only study of a complete set of serial sections of the nervous system ever made in Friedreich's disease. He found diffuse sclerotic plaques in the brain and cord, and he has brought forward once more the French view that the disease is a special form of multiple sclerosis. Rombach thinks that in this case studied microscopically by Brouwer the original diagnosis of Friedreich's ataxy should be changed to disseminated sclerosis. He thinks that the most reasonable view to take is that hereditary ataxy can occur under many various clinical forms; that in the middle stands the sharply defined type indicated by Friedreich (sometimes even very closely related to the "juvenile ataxy" of Winkler and Jacobi), and that on the other side aberrant forms occur in which necropsy reveals a combined systemic disease as well as a cerebellar atrophy. Further, it is found that the whole nervous system is too small while there is no question of any degenerative processes therein. Moreover, it is probable that a cerebellar atrophy without any spinal cord affection can occur as an analogous clinical picture. [LEONARD J. KIDD, London, England.]

Lloyd, James Henry, and Newcomer, Harry S. A CASE OF FRIEDREICH'S ATAXIA. [Archives of Neurology and Psychiatry, August, 1921, pp. 157-162.]

The authors remark that Friedreich's ataxia is still a rarity, and the opportunity to examine the spinal cord in an early stage of the disease does not often present itself. This opportunity occurs in the case of a boy in Lloyd's service at the Philada General Hospital. The patient, aged eleven years, died of an acute influenzal pneumonia. He had a younger brother who also had this familial affection. They were negroes, in which race Friedreich's ataxia is probably even more rare than in the white race.

The patient had had convulsions in infancy. The ataxia began when he was eight. He kept his feet far apart, swayed violently, and required assistance in walking. It was not the gait of tabes. He had the Romberg sign. Scoliosis was present. The knee and Achilles reflexes were absent, and there was no Babinski reflex. Superficial sensation in all its modes was normal, but there was inability to recognize a tuning fork held on the radius, ulna or tibia. No pain, no muscular atrophy, no girdle sense, no loss of sphincter control. Speech was hesitating and staccato, rather than drawling. Vision was normal, the pupils equal, the reaction to light sluggish, the action on accommodation prompt. There was some

loss of pigmentation in areas of the iris. Nystagmus was not present. The disks were somewhat atrophic. The symptoms in the case of the younger brother were very similar.

Wassermann tests were made on the blood of the mother and an older daughter, and on the blood and spinal fluid of the two brothers. They were all negative. The family history was unimportant; no ataxia in any collateral branches.

The authors call attention to the fact that speech defects and nystagmus were not marked in these boys, but these symptoms are sometimes rather late in appearing in Friedreich's ataxia.

Newcomer made a careful study of the histological changes. There was pigmentation of the nerve cells, and a gliosis of the gray matter of the cortex and especially of the cord. There was a tract gliosis involving almost completely the fibers in the posterior columns having the position of the third myelogenetic system, and also involving slightly the direct cerebellar and crossed pyramidal tracts. Also extensive posterior nerve root degeneration. Degeneration did not show clearly in the restiform body, and there was nothing abnormal in the appearance of the dorsal portion of the superior worm, nor any gliosis of this or any other parts of the cerebellar cortex. Normal cells were absent in Clark's columns in numerous sections of the cord. The gliosis in the regions of the greatest changes consisted of a dense fibrous mat—a mat of whorls and irregularly arranged glia fibers. There were some spider cells. Microphotographs of various sections of the cord are given. These show the distribution of the lesions, referred to, in the posterior columns. [Author's abstract.]

Sundberg, C. G. SENSIBILITY IN FRIEDREICH'S DISEASE. [Upsala Läkareförenings Föreläsningar, Sept. 1, 1921, 26, No. 5-6.]

Sensibility in Friedreich's disease—Sundberg gives a detailed account (in English) of the sensory disturbances which were recorded in all of the ten cases of Friedreich's disease analyzed.

Taddei. TRANSITIONAL FORMS OF HEREDITARY ATAXIA. [Revista Critica di Clinica Medica, May, 1921, 22, No. 15.]

This is a clinical report of a young man who inherited neurosyphilis and who showed a typical syndrome of Friedreich's ataxia and some of the symptoms of Marie's hereditary cerebellar ataxia. The Friedreich and the Marie types must thus be regarded as different trends of a fundamental process, not as entities.

van der Loo, C. A. A CASE OF FRIEDREICH'S ATAXY. [Nederlandsch Tijdschr. voor Geneeskunde, 1921, LXV, H. 1, p. 469.]

The writer reports to the Rotterdam Clinical Society a case of Friedreich's ataxy in a youth of twenty. Gait disturbances began at age of eleven, and the ataxy became steadily worse and continuous. Later, he

had influenza. No other illness, and no evidence of lues. Possibly the influenza aggravated the ataxy. He now has slight nystagmus to both sides, weakness of leg muscles but no spasticity, very lively knee- and ankle-jerks, bilateral Babinski sign, thigh and foot clonus, bilateral pes cavus, and extreme ataxy so that he can hardly stand. Arms and hands very ataxic. Definite adiadochokinesis. Pupils normal. No speech disturbance. Here, then, we have involvement of the cerebellum and the spino-cerebellar tracts, and clinically a combination of Friedreich's ataxy and Marie's heredo-cerebellar ataxy. [LEONARD J. KIDD, London, England.]

Noica. THE FIXATION FUNCTION OF THE CEREBELLUM. [*Revue Neurologique*, February, 1921.]

The function of the cerebellum in relation to voluntary movement is here presented in a new manner and the author illustrates his point with a carefully studied case of unilateral lesion of the cerebellum. Voluntary movements demand fixation of the body or portions thereof adjoining the part to be moved. He believes that one of the important functions of the cerebellum is to bring about that fixation. It is known, for instance, that a patient with a cerebellar lesion cannot rapidly pronate and supinate the forearm (adiadochokinesia). Now, Noica notes that if this patient's shoulder and upper arm be fixed against the back of a chair or his side, he can perform the movement without difficulty. Similarly, he may be able to draw circles on a blackboard if the trunk be fixed. He can handle a spoon, fork, etc., if the elbow be held. In many other ways the inco-ordination of movement may be overcome. Hence adiadochokinesia is an indication of loss of ability to fix other parts upon whose stability the wished for movements depend.

Simonelli, G. DEFICIENT POSTURAL ACTIVITY IN CEREBELLAR DISEASE. [*Riv. crit. di clin. med.*, 1921, 22, 3. *Med. Science.*]

The indeterminate character and lack of precision observed in the movements of patients suffering from cerebellar disease are stated to be due, to some extent at least, to imperfect tonic fixation on the part of certain groups of muscles, whereby the limb or limb-segment is deprived of a firm "point d'appui" from which to act. This view has been recently supported by Noica (*Revue Neurologique*, 1921, 28, 164) in a series of clinical observations, and in two cases recorded by Simonelli. In one of Simonelli's patients both upper and lower limbs were affected; in the other mainly the lower limb. The phenomena observed by him were as follows:

1. Patient standing: Directed to touch the tip of the nose, the aim was misdirected on to the cheek and the act was accompanied with extensive movements of shoulder, trunk, and pelvis.
2. Patient seated, with one arm hanging free: Aim misdirected and accompanied with movements of the shoulder and oscillation of the trunk.

3. Patient seated, with the trunk supported by one hand resting on the bed: Aim more precise.

4. Patient seated, with trunk and shoulders fixed: Aim more precise and, after the first attempt, approximately correct.

5. Test for dysadiadochokinesis with arm raised: Movement slow and incomplete, and propagated to the arm and shoulder, so that considerable force was required, on the part of the observer, to steady the arm and shoulder, all segments of the patient's limb apparently participating in the movements.

6. The same test, with the elbow fixed to a firm support: Movements more complete and rapid.

7. Walking: In addition to the characteristic features depending on hypermetria and asynergia, movements of the pelvis and irregular oscillations of the trunk were observed.

8. Patient in dorsal decubitus, with leg raised: Directed to make rapid movements of adduction and abduction of the foot, irregular movements of the thigh and pelvis resulted, rendering execution of the directed movements impossible.

9. The same, but with the leg resting on the bed: The alternate movements of the foot were effected slowly and accompanied with alternate rotatory movements of the thigh, which could be prevented only by the use of much force on the part of the observer.

10. Patient seated, with the heel resting on the floor and the thigh fixed by the observer's hands: The alternate movements of the foot were effected in a fairly normal manner.

In an abstract by the author himself the following points are developed:

The phenomena of cerebellar insufficiency observed in experimental animals following total and partial destruction of the organ consist especially in deficiency of muscular tone. In the spontaneous affections of the human cerebellum the symptom of atonia has not been given great importance up to this time, because the clinicians have been struck by the more complex symptoms such as the disturbance of equilibrium, asynergia, adiadochokinesia, etc. The spontaneous lesions in fact consist in a variety of very complicated pathological processes which reveal their action progressively and involve the functions even of remote organs. Circumscribed lesions of the cerebellum, in every respect comparable to those of experimental surgery, have appeared in the injuries of the war. In these cases A. Thomas and G. Holmes have plainly shown the importance of tonic insufficiency. But according to the view of Sherrington the tonus has a complex function, defined by this author as "postural activity," destined to maintain the static postures of the body and to exert an opportune stabilizing action upon the various skeletal segments. This conception aids greatly in interpreting certain cerebellar symptoms because, as G. Rossi has already observed, one part of the

disturbances of motor ability may be considered as dependent upon the insufficiency of the postural tone. In fact in order that one segment of the body shall move regularly it is necessary that a stable point of articulation should be found in a neighboring segment and that all parts of the result are harmoniously joined. Thus the fine movements of the finger presuppose especially the stabilization of the articulation of the wrist. Those of the forearm presuppose the stabilization of the arm and of the shoulder and require the firmness of the hand.

Noica, studying some clinical cases, has taken into consideration the importance which the cerebellum has in the regulation, to which he has attributed "a rôle of fixity in the execution of the voluntary movements." This "rôle of fixity," according to Simonelli, is only one way of expressing the "postural activity" of Sherrington.

Simonelli compares these theoretical considerations with the results obtained from his own observations in two clinical cases. These were two old cerebellar cases in which some symptoms, hypermetria, adiadochokinesia, notably had corrected themselves or disappeared in the upper as well as in the lower extremity, when the deficient fixating action of definite groups of muscles was artificially corrected. Thus the test of touching the tip of the nose with the index finger became more correctly performed when the trunk and the shoulder were immobilized; the adiadochokinesia of the hand was diminished most when the forearm was fixed; that of the foot, abduction, adduction, when the hip was fixed and the heel made to touch the ground. The author, moreover, has observed that the proximal segments to the one that should move not only do not remain fixed but perform energetic active movements. According to author this is a phenomenon of functional adaptation, a substitution of clonic action for the tonic action which is absent, designed to facilitate the movement of the distal segment. [Author's abstract.]

Steuber, P. J. SARCOMA OF CEREBELLUM. [Ohio State Med. Jour., May, 1920.]

A clinical report of a sarcoma of the cerebellum in a boy of eight years who had been sick a year. At the necropsy the cerebellum presented on section a sharply circumscribed, necrotic, caseated mass, replacing practically all the entire vermis, and extending well out into the cerebellar hemispheres, leaving only a small capsule of cerebellar structure varying in width from a few millimeters to 2 centimeters. The pathologic diagnosis was small round cell sarcoma.

Paulian, D. E. PYRAMIDO-CEREBELLAR DYSGENESIS OF A FAMILIAL TYPE. [Bucarest, 1921.]

Familial pyramido-cerebellar dysgenesis is the term applied by the author to the peculiar condition observed in a brother and sister nine years previously in Marinesco's clinic simulating multiple sclerosis. The girl gives the following history: At the age of twenty years onset of

the disease with tingling and swelling of the right arm and weakness of the lower limbs, the gait becoming more and more unsteady. Within four years it had advanced so that she could only drag the left leg when supported, and she became bedridden. There were sphincter disturbances, retention, then incontinence of urine, internal strabismus of the left eye, pupils equal and no nystagmus, parasthesias of both legs, loss of muscular power of the flexors of both legs, bilateral Babinski reflex, ankle and patellar clonus, exaggerated tendon reflexes generally of both legs, asteriognosis, dysmetria, intention tremors in both hands, bilateral adiadochokinesis, slow, syllabic utterance. Wassermann reaction of the spinal fluid negative. No lymphocytosis. The brother was in good health until he reached the age of twenty, when he complained of numbness in both legs, no pain, unsteady gait. On examination he was found to have exaggerated tendon reflexes, bilateral Babinski, bilateral clonus, intention tremor of both hands, positive Romberg, ataxia, sympathetic flexion, dysmetria, adiadochokinesis, sphincter trouble, retention of urine, sexual impotence. The pupils were equal. Speech slow and indistinct. Negative Wassermann reaction of the spinal fluid. No lymphocytosis. The onset at the same age, and in the same manner is suggestive of the same malady. The mother had syphilis, with several miscarriages, and lost many of her children in their infancy. These two cases resemble Pierre Marie's hereditary cerebellar ataxia, though the characteristic feature—the heredity—is lacking. The authors are more inclined to think that it is a familial type of disease, determined possibly by hereditary syphilis, transmitted by the mother. [Author's abstract.]

Bakker, S. P. NEURITIS AND POLIOENCEPHALITIS SUPERIOR IN TERTIAN MALARIA. [*Nederlandsch Tijdschr. voor Geneeskunde*, 1921, LXV, Feb. 5, 745.]

Bakker reports a case of neuritis and polioencephalitis superior in tertian malaria in a man aged twenty. Early in June, 1920, he had a sudden malarial attack, followed by several more. He improved at first under quinine, and resumed work in a fortnight. In mid-July return of attacks; he had to keep to bed for two or three weeks. On getting up, was very weak in legs, with paresthesia there, and later on hyperesthesia. At first he could not stand; later, he had a drunken gait. About August 12 he had diplopia, and a few days later complete bilateral ophthalmoplegia. On August 18 diminished visual acuteness, ptosis, no ocular movement, no diplopia, pupils acting to light, and normal fundi. Two days later, diminished pupil-reaction and complete palsy of accommodation; slight sensory changes in lower extremities with definite ataxia; ankle jerks very sluggish; spleen not definitely enlarged. Wassermann negative in blood and spinal fluid; Nonne reaction +, and pleocytosis 91 in spinal fluid. A few days later more attacks, and plasmodia, apparently tertian, found in blood. By mid-October the ankle-jerks were absent, and there was still some imperfection of upward ocular movements. Bakker regards

the ataxia, the sensory changes in the legs, and the unequal sluggish ankle-jerks as due to a malarial neuritis. This condition, seen usually in the tropics and only very rarely in Holland, there is often sensory change with little or no motor paresis; according to Schupfer, the R.D. does not occur. Fatal cases have been recorded, with thickening of nerve trunks, swollen varicose medullary sheaths, and loss of axis cylinders. As to the ophthalmoplegia and cycloplegia in Bakker's case: peripheral ocular palsies have been seen in malaria, but in his case a peripheral lesion was unlikely because of the complete symmetry of the signs, the presence of diplopia only in the first days of the illness, the residue left of defective upward movement, and the presence of bilateral rotatory nystagmus. Among central affections of the nervous system in malaria are meningitis, hemiplegia, monoplegia, myelitis, bulbar and cerebellar symptoms, epilepsy, pseudo-tumor cerebri, and paraplegia. Encephalitic foci have often been found, especially subcortical. Von Monakow describes a case of total ophthalmoplegia with a focus in the parietal lobe. Bakker's case is almost certainly not to be localized in the cortex cerebri, still less in the internal capsule. He localized the lesion in or near the nuclei of the ocular nerves, *i.e.*, the case is one of superior polioencephalitis. Bakker shows that the case is certainly not one of lethargic encephalitis. The characteristic of this polioencephalitis superior is always the irregularity and often the symmetry of the ocular palsies, without other signs. Not only was there in this case a toxin at work, but also an inflammation, as witnessed by the very great pleocytosis of the spinal fluid. Bakker finds only one other case of polioencephalitis superior in malaria, *viz.*, that of Ziemann, who, however, does not describe any particular points about it. [LEONARD J. KIDD, London, England.]

Söderbergh, G. CEREBELLAR SYMPTOMATOLOGY. [Acta Medica Scandinavica, Nov. 27, 1920, No. 54, 2.]

If the arms be extended horizontally they were bent involuntarily and rhythmically at the elbow, or twisted to and fro, when the arms were in certain planes and not in others in three cases observed by this author. The movements were most pronounced when the arms were forced in the extreme adduction plane.

Tinel, M. J. PARKINSONIAN SYNDROMES FROM MESENCEPHALIC LESIONS. [Revue Neurologique, July, 1920.]

Two important points concerning Parkinsonian syndromes are associated with lesions in the midbrain. First, there is a dissociation between the slow and the rapid movements. In certain cases there is a contrast between the difficulty of executing slow movements and the ease of executing rapid movements. Of this he has observed two good examples. One of his patients, only able to walk very slowly and supported on both sides, was capable at certain times of taking long strides. The other, able to walk with great difficulty on a level surface, could ascend stairs easily

and quickly and could even go up two steps at a time. This is a paradox, having nothing in common with festination and it is difficult to explain. This phenomenon was observed by Frederick Tilney in 1911, who spoke of it as "metadromic progression." Secondly, there is a difference between the dynamic and the static force, one which has been noted since the time of Trousseau and has been particularly studied by Dyleff. But Tinel thinks that this has no Parkinsonian or even pathological character, but is simply a normal phenomenon.

Hunt, J. Ramsay. THE STATIC OR POSTURE SYSTEM AND ITS RELATION TO POSTURAL HYPERTONIC STATES OF THE SKELETAL MUSCLES, SPASTICITY, RIGIDITY AND TONIC SPASM. [Neurological Bulletin, 3, 1921, p. 207.]

According to the author, motility is subserved by two components, each represented throughout the entire efferent nervous system by separate neural mechanisms, which are physiologically and anatomically distinct.

One component is the movement proper, which is subserved by the *kinetic system* (motion system). The other represents that more passive form of contractility which we recognize in tonus, posture, attitude and equilibrium. This posturing or static function of the efferent system is, he believes, subserved by separate neuromuscular pathways, the *static system* (posture system).

The term static is used here to designate that peculiar property of the muscle fiber by which it becomes fixed in posture. In the reflex nervous system this is manifested by postural tonus and at the higher levels by the various postures and attitudes. There are many reasons for the assumption that the kinetic system is related to the anisotropic discs and the static system to the sarcoplasm of the muscle fiber as these elements differ in structure, innervation, mode of contractility and metabolism. There are also reasons for the assumption that the transformation of movement into posture is effected by fixation of the sarcoplasm which thus converts the contractile muscle fiber from a kinetic into a static mechanism.

According to this conception, then, movement is subserved by a kinetic, and posture by a static mechanism, the two systems supplementing one another and working together in harmony. For every movement starts from posture, is accompanied by posture, and terminates in posture, posture following movement like a shadow. At the same time, the postural mechanism would exercise a stabilizing and steadying influence upon the course of movement itself.

The kinetic system is concerned with the active movement; the static system is concerned with what might be termed the passive form of movement as expressed in postures and equilibrium. Both are manifestations of the efferent mechanism but differ in the character of their function, although subserving a common end—motility. Motility as it is observed in animal life may be divided more or less arbitrarily into three distinct

groups, viz., reflex, automatic-associated, and isolated-synergic or dissociated types of movements.

All of these forms of movement are intimately associated with that other component of motility which we term posture. And the same postural groups may be recognized as in the classification of movement, viz., *reflex postures*, *automatic-associated postures* and *isolated-synergic types of posture* corresponding to their respective motor mechanisms. And it may be stated as a general principle that there is no form of motility from the simple reflex to the most skilled and individualistic type of movement which functions without a corresponding static or posture mechanism.

Anatomical Considerations—In a general way, the central nervous system may be said to represent three great structural and functional divisions. There is the segmental nervous system which contains the great reflex systems of the neuraxis, and is the archaic representation in man of nervous reactions of the lowest forms of life. The other is the paleo-encephalon represented by precursors of the corpus striatum and optic thalamus which constitute the higher coördinating sensory and motor mechanisms of the lower forms of life. The neo-encephalon represents the latest stage in the evolution of the nervous system and has its greatest development in man. These three great functional divisions of the nervous system are related to the three great types of movement and posture already mentioned. The essential integrating and correlating mechanism for the control of static contractility he believes to be the cerebellum. Afferent impulses from the periphery and efferent impulses from the cerebral cortex pass to this organ before their final distribution by way of the cerebello-spinal system to the posturing or static mechanism of skeletal muscles. The cerebellum presents phylogenetic evidences of two great divisions, a paleo-cerebellum and a neo-cerebellum, which correspond to similar divisions of the cerebrum. The paleo-cerebellum receives impulses directly from the spinal cord, which pass to its central portion and constitute the vermician system. The neo-cerebellum receives impulses from the cerebral cortex, which pass by way of the pons varolii to the cerebellar hemispheres, and constitute the hemispheric system.

The older cerebellum stands in relation to what may be termed the *paleostatic system* which controls the older static or postural functions of "automatic and associated type." It takes its origin in the older nuclei of the vermis cerebelli (N. Fastigii, globosus, emboliformis). The automatic-associated posture of standing, as observed in decerebrate rigidity, would be one of the functions of this paleostatic system.

The cerebellar hemispheres, on the other hand, regulate the higher postural functions of motility through the medium of a *neostatic system*, which takes its origin in the cells of the dentate nucleus. The neostatic system is controlled from the cerebral cortex by special tracts which connect the various regions of the cerebral cortex (frontal, parietal,

temporal and occipital) with the dorsal and ventral nuclei of the pons varolii, and thence to the opposite hemisphere of the cerebellum.

In posture, as in movement, no sharp line of demarcation can be drawn between the various types, and one group merges imperceptibly into another. It is probable, he believes, on phylogenetic grounds, that the parieto-temporo-occipital tract to the dorsal pontine nuclei is older than the fronto-pontine system which descends from the frontal lobe to the ventral nuclei of the pons. The latter system he regards as the homologue in the static or posture sphere of the cortico-spinal or neokinetic system. The fronto-ponto-cerebellar (*neostatic system*) descends in the anterior limb of the internal capsule and mesial portion of the cerebral peduncle in close relationship with the pyramidal tracts, and it is interesting to note that both structures, the one subserving the neostatic and the other the neokinetic functions of motility, receive their myelin sheaths subsequent to birth.

The other cortico-cerebellar systems (parieto-occipito-temporal systems) pass in the lateral portion of the crus cerebri to the pontile nuclei, and thence to the opposite cerebellar hemisphere. It is his belief that they represent cortico-cerebellar connections subserving higher automatic associated types of posture in connection with the various sensory areas of the cerebral cortex and are the homologues in the static sphere of cortico-thalamic connections with the strio-spinal or paleokinetic system. It is interesting to observe that all the posture systems, *neostatic* as well as *paleostatic*, pass to the cerebellum for final integration and coördination. This is in accord with the nature of the posturing mechanism and its secondary and unconscious rôle in motility. For while the higher forms of movement are initiated as conscious and voluntary processes, the corresponding postures are secondary, and follow automatically in the path of movement.

The Static System and Postural Hypertonicity of the Skeletal Muscles—As we have seen, there are as many types of posture as there are of movement, and with corresponding development of neural mechanisms. We must therefore recognize the existence of special systems of fibers in the cortical, capsular, crural, cerebellar, pontile bulbar, spinal and peripheral divisions of the nervous system to carry out this static or postural function, corresponding to homologous systems for the kinetic or mortorial function.

When some portion of the kinetic system is injured, paralysis immediately results; if the injury takes place in the central nervous system the lower motor centers are released from control, and a characteristic postural rigidity develops. This peculiar overaction of certain muscles takes place in response to the principle that destruction of a higher center releases the lower kinetic mechanisms from control. When this has taken place, as after all other forms of movement, the motion-complex becomes fixed in posture and we have produced that curious paradox of a para-

lyzed center above and a functioning center below, which has expressed itself in terms of *postural-hypertonus*.

Types of Post-Paralytic Postural Hypertonicity—Many different types of postural hypertonus following paralysis may be recognized, which depend upon the nature and localization of the lesion within the central nervous system. The more important of these are the spasticity of pyramidal tract disease; the muscular rigidity of paralysis agitans; the decerebrate rigidity of midbrain section, and the postural hypertonic effects of transverse lesions of the pons, medulla and spinal cord.

Cortical or Pyramidal Types of Postural Hypertonus (spasticity)—One of the most characteristic symptoms of organic disease of the central nervous system is spasticity, or spastic hypertonicity of the skeletal muscles. The affected muscles while weak or paralyzed are in a state of overaction and postural fixation which is dependent upon the overactivity of subordinate kinetic and static centers. Any injury of the neokinetic or corticospinal system is followed by paralysis of the isolated-synergic movements of cortical origin and release of the lower motor mechanisms of the spinal cord. This is commonly referred to as a loss of cerebral inhibition. In spasticity the kinetic and the static systems are both in action. There is a kinetic discharge which is accompanied by postural fixation.

Striatal or Pallidal Types of Posture Hypertonus (paralysis agitans rigidity)—The rigidity of paralysis agitans is a postural hypertonicity resulting from paralysis of the pallidal system of the striospinal pathway. In previous studies of paralysis agitans this point of view has been presented, together with evidences showing that the efferent motor system of the corpus striatum (pallidal system) represents an internuncial common pathway for the passage of motor impulses for automatic-associated types of movement (paleokinetic system). The characteristic attitude and postural deformities of this affection present many points of difference from those associated with the spastic state. There is the mask-like expression of the face, the peculiar attitude of head, trunk and arms, and the characteristic position of the hand and fingers. The distribution of the palsy and associated muscular rigidity differ essentially from that observed in the spastic state.

Pallido-Pyramidal Types of Postural Hypertonus (spastic rigidity)—The proximity of the corpus striatum (pallidal system) to the internal capsule (pyramidal system) is such that simultaneous involvement of both structures is not uncommon. Indeed, in vascular lesions of the basal ganglia, it is probable that pure forms of either pyramidal or pallidal palsy are quite rare and that a combination of the two types is the more common.

Involvement of both systems, the pallidal and pyramidal, produces a *spastic-rigid state* which combines in varying degrees the characteristics of both types of postural rigidity.

Midbrain or Paleocerebellar Type of Postural Hypertonus (decerebrate rigidity)—Another type of hypertonicity which has awakened considerable interest and investigation, is the decerebrate rigidity of Sherrington. This is a peculiar state of muscular rigidity which corresponds to the automatic-associated posture of standing and is produced by a section through the midbrain at the level of the anterior colliculi. A mesencephalic transection at this level severs the pyramidal tracts (neokinetic system), the pallidal tracts (paleokinetic system), as well as the cortico-ponto-cerebellar tracts (neostatic system). The various subordinate centers of the pons, medulla and cord, both kinetic and static, are released and the skeletal muscles assume the postural rigidity of standing which is characteristic of this level. As the older static system of the cerebellum (paleocerebello-rubro-spinal system) for the control of automatic-associated types of posture is still functioning, retaining its afferent connections with the spinal cord and the efferent with the red nucleus, the automatic posture of standing, characteristic of decerebrate rigidity, is maintained. When the section is carried below the red nucleus, which severs the paleostatic system, the typical decerebrate posture no longer appears and the other types of postural reaction make their appearance (pontine and bulbar). Decerebrate posture is also abolished by section of the superior peduncles of the cerebellum, which would sever the connection between the efferent cerebellar system and the red nucleus. Decerebrate rigidity may, therefore, be regarded as the postural expression of paleostatic activity, which develops after the elimination of cortical and striatal inhibition.

Spinal Types of Postural Hypertonus (paraplegia in flexion)—A transverse lesion of the cord would sever all of the long projection systems, both static and kinetic, and release its various intrinsic spinal reflex mechanisms. In response to this loss of the higher inhibitory control, the characteristic reflex of flexion develops which becomes fixed in postural hypertonus, the extreme expression of which is "paraplegia in flexion." An extensor type of spinal paralysis is also encountered (paraplegia in extension). This form is characteristic of pyramidal tract disease and is merely the spinal expression of the *pyramidal* type of hypertonus, as observed in purely spastic states. The author, in conclusion, reiterates that the function of motility is subserved by two distinct components, which express themselves in movement and in posture, and which are served by a kinetic or motion system and a static or posture system. Both systems are represented throughout, from the lowest to the highest level of the efferent neuro-muscular mechanism. The reflex nervous system is concerned with reflex movement and reflex posture, the paleokinetic and paleostatic nervous system with automatic-associated types of movement and posture, and the neokinetic and neostatic with isolated synergic types of movement and posture. The essential correlating mechanism of all static or postural activity is centered in the cerebellum, which is under the control of spino-cerebellar and cortico-

cerebellar pathways. There are two forms of cerebellar activity, paleo-cerebellar and neocerebellar, which correspond to different types of posture. The neostatic function of the cerebellum is under the control of a cortico-ponto-cerebellar system (fronto-pontine tracts). The paleostatic function of the cerebellum is under the control of the spino-cerebellar system. According to this conception the static system, like the kinetic, is represented in the central nervous system by neostatic and paleostatic systems which subserve the older and more recently acquired forms of posture. This is in harmony with the author's theory that motion and posture develop side by side as twin mechanisms. When there is movement there is also posture, and when a kinetic mechanism is formed, a static mechanism is also formed, a parallelism which may be traced in the whole field of motility both functional and structural. All of the various postparalytic types of postural hypertonus are indications of overactivity in subordinate motion and posture centers following paralysis and loss of the higher mechanism. [Author's abstract.]

Hirose, K. THE NUCLEUS IN THE HUMAN RESTIFORM BODY. [Bull. Johns Hopkins Hospital, 1921, XXXII, October, 336 (12 figs.).]

In thirteen human brains of both sexes, both foetal and adult, Hirose has found a special nucleus in the restiform body. He failed to find it in several other mammals specially examined for it. In transverse sections from below upwards we first see it where the vago-glossopharyngeal root begins to disappear, and it ends where the accessory auditory nucleus begins to appear. It is found at the dorso-medial part of the restiform body on transverse sections, and it extends in an elongated shape in the bulb. It has no definite shape, and shows much variation in growth. Some transverse sections show a hilum tending towards the medial direction. The nerve cells of the nucleus are practically identical in shape with those of the inferior olive. Hirose thinks the nucleus corporis restiforme is probably a portion of the inferior olive; he says that, as far as he knows, it is undescribed in the literature (but Jergelsma describes it as present in the cat [*Journ. für Psychol. und Neurol.*, 1917, XXIII]; for he found in three cases of cerebellar atrophy in cats—in which the atrophy was especially prominent in the zona glandulosa of the cerebellar cortex—that the cells of the pons, nuclei literales medullæ, and the nuclei corporis restiforme were considerably altered, but that the inferior olivary cells were completely normal; this would indicate that in the cat there is a restiform-cerebellar tract whose fibers end in the zona granulosa of the cerebellar cortex). [LEONARD J. KIDD, London, England.]

Bergman, E. HEREDITARY ATAXIA. [Upsala Läk. Förh., September, 1921, 26, No. 5-6.]

In this clinical study three families with Friedreich's spinal ataxia and one with Marie's cerebellar ataxia are presented by the author, with complete genealogical data. In the Friedreich cases signs of family

inferiority are common, but in the Marie case the family stock is good. In none of the families is there a history of syphilis or abuse of alcohol. In one of the families with Friedreich's ataxia, six of the fifteen children present it; in one other family four of the eleven children. In none of these three families were other cases of the ataxia known in parents or grandparents. In the Marie family there was one member affected with the cerebellar ataxia in three generations, and no other instances were known in the other generations or among the twenty-one other members of the four families descended from the one couple in the second generation. Besides the fourteen patients with pronounced spinal or cerebellar ataxia in these families, Bergman gives the details of the other members of the families who present signs of degeneracy although free from ataxia. The article is in English.

Casaubon and Muneagurria. FRIEDREICH'S DISEASE. [Arch. Lat.-Am. d. Ped., January, 1922, 16, No. 1.]

An isolated instance occurring in a boy of seven with syphilitic taint without familial involvement, and following an attack of diphtheria.

Winkler, C. THE HUMAN NEO-CEREBELLUM. [Br. M. J., May 13, 1922.]

An exchange lecture arranged by the University of London was delivered at the Royal Society of Medicine on May 3 by C. Winkler, professor of clinical psychiatry in the University of Utrecht, who took for his subject the human neo-cerebellum. Mott, who took the chair, welcomed the lecturer as one whose neurological work was known all over the world, and who was director of an institution which could be regarded as a model of its kind.

The lecturer first paid a tribute to the British school of neurology and physiology for their work on the functions of the cerebellum, and especially to the memory of Hughlings Jackson, whose clear views on cerebellar functions came as a revelation to his contemporaries.

Winkler introduced the subject of his lecture by an account of a case of olivo-ponto-cerebellar atrophy; the account was illustrated by lantern slides showing the condition found *post mortem*. The patient was a man who was under Professor Winkler's observation for five years, and died at the age of fifty-nine; one brother was similarly affected and died of the same disease. When the patient was first seen the regular symptoms of cerebellar ataxy were present. At the age of fifty the patient had complained of dizziness on moving his head up and down, and at the age of fifty-two he had the staggering gait of a drunken man. In a very short time his speech became affected. At the age of fifty-three the patient presented the following characteristic signs and symptoms:

1. *Dysmetria* in all his movements was marked. He could not touch a defined spot with his blackened forefinger. He always pointed with his right forefinger above and to the right, and with the left above and to the left. In later years his movements deviated in other directions.

2. *Dyssynergia*.—On moving his trunk backward he fell with extended legs, never making flexion of the knees. Later all his movements became helpless. When put on his legs he moved the trunk to the left, and trampled desperately with his legs in the air.

3. The upper limbs were very atonic, and there was marked dysadiadokokinesis on both sides. It was difficult to estimate the tonus in the lower limbs. The knee and Achilles reflexes were exaggerated. Babinski's sign was never observed.

4. He spoke as if a potato were in his mouth, which made the words unintelligible, and a kind of gibberish.

5. Nystagmus was never observed.

6. He responded well to all tests of touch, pain, and temperature.

7. Vision was bad owing to strong myopia caused by staphyloma posticum without optic atrophy.

8. In the earlier stages hearing was good and the vestibular reflexes were normal. During the last year of his life he became deaf.

He died of pneumonia at the age of fifty-nine.

At the autopsy the brain weighed 1,380 grams—a good average weight. There was no affection of the vessels or membranes and nothing abnormal noticeable, except a small pons, a failure of prominence of the olivary bodies, and a smaller cerebellum than normal. Serial sections of the pons and medulla with the cerebellum were made and stained by the usual methods to show the conditions of the cells and fibers. A marked abiotrophy of the pontine, olivary, and arcuate nuclei was demonstrated in Nissl-stained sections; the other nuclei were intact. As a result of this neurone atrophy certain fiber systems were seen to be absent when compared with the normal.

These facts were demonstrated by means of Weigert-stained specimens projected by the lantern. Thus the inner portion of the inferior peduncle, which consists of fibers from the inferior olive and arcuate fibers, was sclerosed, and a very great falling out of fibers from the pons to the middle peduncle was obvious. Consequently, there was compared with the normal a great loss of fibers in the medullary rays of the cerebellum, but around the dentate nucleus the white matter was well stained, showing that the fibers entering this nucleus were not atrophied. The superior peduncle showed no degeneration. Whereas there was great loss of fibers in the plexus of the granule layer of all the lamellæ, the fibers around the Purkinje cells showed a well-marked pericellular fiber plexus. The climbing fibers of the spinal tracts of Gowers and Flechsig have their terminal arborizations in this plexus.

Granted, then, that there was a primary systemic abiotrophy of neurones having a definite destination to the whole of the lateral lobes of the cerebellum, it must, the lecturer said, be asked, What was its functional and biological significance? The nature of the problem became more apparent when these facts were correlated with the results

of experimental lesions in animals and embryological investigations on the development of the nervous system in the human subject.

Winkler mentioned the effects of experimental removal of the cerebellum in animals, and showed sections in which the flocculus on one side had not been extirpated. He concluded from a study of Marchi preparations that as long as the layer of granules persisted there was no loss of cells in the nuclei connected with it. The argument he adduced was that the loss of cells in the pontine, olivary, and arcuate nuclei was not consecutive to an atrophied cerebellum in which the layer of granules showed only slight alterations, as was found in the case he described. His view was, therefore, that the cerebellar atrophy which he found was due to an atrophy of the medullary rays the result of a primary atrophy of the pontine, olivary, and arcuate nuclei. The lecturer then referred to the researches of Essick upon the human foetus of $2\frac{1}{2}$ cm., in which he showed that two bands of neuroblasts (the bands of Essick) exist—the one, distal, surrounding the bulb laterally, and forming later the olivary and arcuate nuclei; and the other, the proximal band which forms the pontine nuclei.

Winkler expressed the view that these olivo-arcuate-pontine cells, being of later phylogenesis, have not the same vital energy to resist degeneration as is possessed by the evolutionally older cell structures, and are therefore more susceptible to abiotrophy. He considers that these cases of primary olivo-arcuate-pontine degeneration support the embryological observations of Essick, which indicate that these structures are of later evolutionary development. Moreover, the olivo-arcuate fibers in the inferior peduncle are myelinated considerably later than the spino-cerebellar fibers. This was demonstrated by specimens stained by Weigert's method.

Upon these grounds Winkler is of opinion that the views of Edinger and Conolly—based upon studies in comparative anatomy—that the flocculus and vermis constituted the (old) palaeo-cerebellum, and the lateral lobes the (new) neo-cerebellum, must be modified. There is not in evolution a superimposition of a new cerebellum upon an old cerebellum, but an interpolation of a new into an old structure. He points out that the preponderant importance of the floccular development is undoubtedly lost in the human subject, but that it still retains a fundamental function connected with static equilibrium. He assumes that the olivo-arcuate-pontine fibers being myelinated at a later date are of later evolutionary origin.

Winkler then illustrated by a wax model the evolution of the neo-cerebellum by interpolation instead of by superimposition of a new on an old cerebellum, but, although it was quite possible, he was unable to assert that there was successive myelination of the cortical cerebellar fields which corresponded to the myelination of the spinal, arcuate, olivary, and pontine fibers.

Carp, E. A. D. E. A CASE OF HEREDITARY CEREBELLAR ATAXY.
[Nederland. Tijdschr. voor Geneeskunde, 1922, LXVI, January 21,
p. 350.]

Carp reports a case of hereditary cerebellar ataxy to the South Holland Neurologists' Society. Patient was a man of twenty-five, son of first cousins. No known similar case in family. For three years difficulty in walking. Gait is now slow, wide-based, and of inebriate type. He often falls, especially on sudden movements or on turning round quickly. He has vertigo, but no tinnitus, no diplopia, no diminution of visual fields. *Pari passu*, with the gait-difficulty his speech has deteriorated; it is very quick, explosive, the words run together, and he is often quite unintelligible. As a child, patient was normal, and not at all backward. He was born naturally at full term. The ataxy is greatest in his legs, especially seen in station and locomotion. The reflexes in the lower extremities are increased, with doubtful bilateral Babinski sign; lower limbs definitely hypotonic. Abdominal reflexes present, rather brisker on left side in lower abdomen. Slight scoliosis to left in dorsal vertebral column. There is a regular horizontal nystagmus which increases on looking to left. The eye-grounds and the visual fields are normal, and there is a negative Wassermann reaction in the blood. [LEONARD J. KIDD, London, England.]

Benjamins, C. E. A GRAPHIC METHOD OF RECORDING THE DEVIATION IN THE FINGER-POINTING TEST. [Nederl. Tijdschr. v. Geneeskunde, 1921, July 23, No. 2, 535.]

Benjamins demonstrates to the Netherlands Society for Laryngology, Rhinology, and Otology a graphic method of recording the deviation in the finger-pointing test used in the Italian Air Service during the war. A paper disc, with circles 3 cm. from one another, is fixed to a table, and this was either clasped between the candidate's legs or fixed to the rotation stool by a movable arm; Benjamins prefers the latter, for no new factor (muscle sensibility of legs) is introduced. A little sponge, steeped in various coloring materials, is fixed to the finger; Benjamins moistens the finger tip with water and then rubs it with a water-color block. The Italians soon found that Barany's "normal" rotation-reaction is not constant; according to him, after rotation the patient's outstretched finger should deviate in the direction of the rotation and against that of the after-nystagmus. Five types of reaction were found: (1) Barany's "normal," (2) one that shows no influence of the rotation, (3) an inverted type, in which the hands deviate in the opposite direction from that of the rotation, (4) a diverging type in which the hands diverge from each other, and a converging type in which they may even cross over each other, and (5) an irregular type. Benjamins confirms types 1 to 4, and observes that this has nothing to do with the nature of the after-nystagmus which was in all of normal duration and direction. He concludes that the reaction to the pointing test can be used for diagnosis

only in certain conditions, and that great weight must be attached to the other data. [LEONARD J. KIDD, London, England.]

Zangmeister. OBSTETRIC TEARS OF THE TENTORIUM CEREBELLI.
[Zentralbl. f. Gynak., April 2, 1921.]

Tears of the tentorium are the most frequent cause of intracranial hemorrhage in the newborn. Of 27 consecutive cases, a rent in the tentorium could be demonstrated in 17, and with certainty excluded in 4 only. On the other hand, of 24 such tears demonstrated at autopsy, 6 were unattended by intracranial hemorrhage. In this series the presentation had been: brow 2, breech 3, transverse 2, vertex 17. Forceps had been applied in 5 cases only. The etiological factors concerned are: (1) diminution in the resistance of the foetal skull—for example, from imperfect ossification or premature birth; (2) mechanical obstruction to birth—for example, from contracted pelvis; (3) increased pressure on the head—such as that which follows, for example, from abnormally strong pains. In 22 out of 28 cases birth had been preceded by some degree of intrauterine asphyxia of the foetus, due to prolapse of the umbilical cord, to placenta praevia, or to premature placental detachment.

Walshe, F. M. R. W. ON DISORDERS OF MOVEMENT RESULTING FROM
LOSS OF POSTURAL TONE, WITH SPECIAL REFERENCE TO CEREBELLAR
ATAXY. [Brain, 1921, 44, 539.]

Since the publication by Flourens of his classic researches on the cerebellum a hundred years ago, the functions of this organ have been extensively and earnestly studied both by the experimental and the clinico-pathological methods. While it is not surprising that the complex problems of cerebellar physiology have so far eluded complete solution, it is noteworthy that a symptom-complex so closely and so repeatedly studied as that resulting from destructive lesions of the cerebellum should still remain so variously described and so diversely interpreted.

On the surface, the vast literature which has grown up round the subject shows no common basis of agreement among the various authorities, and the impression derived from it is one of extreme confusion and uncertainty. Two factors appear to be responsible for this state of affairs. The first is the elaborate and empirical nomenclature employed for description, a nomenclature each term of which has been employed in as many conflicting senses as there have been writers. However, as soon as the reader discards this oppressive vocabulary he finds that beneath it there is a very real measure of agreement as to the facts of observation. The second factor, and probably the fundamental one, has been the complete omission by modern thinkers on this problem, to take into account the fruits of modern experimental research on the physiology of movement. The physiological processes underlying the production and coördination of movement and of posture, as revealed by the classic researches of Sherrington, Graham Brown, Magnus and de Kleijn and

others, must form the groundwork of any attempt to understand the functions of the cerebellum, or to analyze the symptom-complex due to destructive lesions of that organ. Nevertheless, by some strange self-denying ordinance students of the cerebellum have not availed themselves of this rich store of fundamental knowledge. The present paper is an attempt to resurvey the problem in the light of the acquisitions to physiological knowledge of the past twenty-five years. If we take such authoritative descriptions of the cerebellar symptom-complex as those of Luciani, Babinski, André-Thomas and Gordon Holmes and, abandoning the nomenclatures adopted, tabulate in simple descriptive terms the several disorders of coördination described we obtain such a classification as the following:

1. Abnormalities of the resting muscle—Loss of tone.
2. Abnormalities of muscular contraction—(i) Excessive range and force of contraction. (ii) Intermittent and unsteady contraction. (iii) Premature relaxation. (iv) Extreme readiness of fatigue.
3. Abnormalities of voluntary movement—Faulty functional combination of the muscles engaged in a voluntary movement.
4. The effects of voluntary efforts at compensation, which are carried out by a musculature functionally defective in the above senses.

It is apparent that this is no final analysis of cerebellar ataxy, for the disorders noted under heading 3 must be in part the natural result of those under heading 2.

A prime necessity is a scientific conception of muscle tone, and of what atonia involves. In clinical literature no such conception is to be found, and the author believes this to be the essential factor in our failure to understand the nature of the problems presented by the cerebellum. He adopts the physiological conception of tone formulated by Sherrington, and he gives an account of the results of abolition of tone on the reflex contraction of striated muscle and on the reflex coördination of movement and of posture, as seen in the experimental animal. In the decerebrate animal, a given muscle may be rendered atonic by section of its afferent nerve supply, while spinal transection also produces a profound, if not total, impairment of tone in the paralyzed muscles. These results may be summarized as including an unduly rapid and excessive contraction with premature relaxation, an unsteady and intermittent contraction, frequent intercurrent inhibition of contraction, a loss of the tonic after-discharge which normally fuses the component contractions of the muscle in any prolonged movement, and extremely rapid fatigue. He observes that these defects include all those found to be the basis of cerebellar ataxy, and he discusses the inevitable results of atonia on coördinated voluntary movement. In conclusion he proposes a unit formula for cerebellar ataxy, namely, a loss of postural tone, or atonia, and he concludes that all the various manifestations of disordered movement seen in a case of cerebellar ataxy are simply particular manifestations of atonia, when this is understood in the physiological sense.

The striking differences of cerebellar ablation in the intact and in the decerebrate animal are then discussed, and it is suggested that the cerebellum is the organ through which the cerebral motor mechanisms regulate posture, of which muscle tone is the basis, and secure the proper coöperation of the phasic and postural components of voluntary movement. [Author's abstract.]

Brouwer, B., and Coenen, L. RESEARCHES ON THE CEREBELLUM. [Psychiatrische en Neurologische Bladen, 1921, No. 3 and 4, May–August, p. 201 (13 figs.).]

The writers discuss three questions connected with the cerebellum: (1) whether there are many intracerebellar association fibers, (2) the origin of the moss-fibers and the climbing fibers of the cerebellum, and (3) the question of localization of speech in the cerebellum. From their experiments on rabbits and from examination of a 42 cm. human foetus and a newly-born child they conclude that the number of intracerebellar association fibers is very great; in the foetus they are specially evident running between flocculus and paraflocculus. From a study of a case of softening strictly limited to the cerebellar cortex they conclude that these intracerebellar fibers rise in a part of the Purkinje cells. Cerebello-corticopetal stimuli arrive partly by the moss-fibers which end round the cells of the zona granulosa, and partly by the climbing fibers which end in the molecular layer. From their study of a case of cerebellar atrophy, in which the chief part of the morbid process—a parenchymatous degeneration of the nerve elements with secondary glia proliferation—had taken place in the molecular layer and in the layer of Purkinje cells, the granular layer being much less affected, the degeneration was confined to the cells of the inferior olives, and the olivo-cerebellar tract was barely perceptible, but the cells of the pons, of the nuclei laterales medulla oblongata, and of the nuclei of the restiform bodies were unchanged. They conclude that the inferior olivary cells send their axons as climbing fibers which end in the cerebellar cortex, whereas the moss-fibers rise in cells of the pons, lateral nuclei of medulla, and restiform nuclei. To determine other sites of origin of the moss and the climbing fibers (spinocerebellar paths, vestibular nuclei, etc.), we must wait for other facts. These results thus differ from Cajal's teaching, and agree with the findings of Jelgersma in some cases of familial atrophy of the cerebellar cortex in cats. On the question of the localization of speech in the cerebellum the writers refer to the current teaching that bilateral cerebellar lesions are necessary to produce the dysarthria that is often seen in cerebellar affections. Recently Stenvers has contended that in unilateral cerebellar lesions speech disturbances can occur, and that in right-handed patients they do so in right-sided cerebellar lesions, and in left-handed in left cerebellar lesions. The writers record a case of a right pontocerebellar angle tumor, in a right-handed patient, which had destroyed the greater part of the right cerebellar hemisphere (and had affected the left slightly) without

giving any speech disturbances during life; they hold that this case does not support Stenvers' teaching. They think that in those cases of unilateral cerebellar lesions in which dysarthria is present this symptom is probably produced by pressure on the contralateral cerebellar hemisphere, and that in the solution of these physiologico-anatomical questions it is better to rely on cases of atrophy of the cerebellar cortex, for here there is no question of pressure on neighboring parts being present. [LEONARD J. KIDD, London, England.]

Parkman, A. CHECKED MOVEMENTS IN CEREBELLAR DISEASE. [Acta Medica Scandinavica, January, 1921, 54, No. 3.]

The diagnostic value of the checking of a movement in cerebellar disease is here discussed. When the finger is rapidly raised to touch the nose, in certain cerebellar disorders, the movement is arrested as if by a check-rein; the rest of the way it can be moved but slowly or jerkily. This symptom is of localizing value when it occurs on one side of the body only.

Klippel and Florand, J. SYNDROME OF SPASMODIC LAUGHTER AND CEREBELLAR TITUBATION. [Presse Médicale, 1921, Nov. 26, 944.]

The patient, as the sequel of an acute illness, probably an attenuated form of lethargic encephalitis, and under the operation of depressing moral causes, showed various nervous symptoms, and especially attacks characterized by (1) spasmodic laughter of subcortical origin, analogous to that of pseudobulbar palsy, coming on without any previous ideas of merriment, and (2) cerebellar titubation. These two kinds of symptoms sometimes come simultaneously, sometimes dissociated; but always they occur in attacks of a few seconds' duration. The patient is on the way to recovery. [LEONARD J. KIDD, London, England.]

Christiansen. DOUBLE THALAMIC SYNDROME. [Rev. Neur., August, 1920, 27, No. 8. J. A. M. A.]

In the case described by Christiansen, the constant pains with violent exacerbations, rebellious to all treatment, even large doses of morphin, are accompanied by choreiform, athetotic and ataxic movements, and there is also dissociation of sensibility, especially in the arms. This thalamus syndrome is bilateral in this case, which is exceptional. It began slowly and insidiously at the age of sixty-three in a man without pathologic antecedents. Pains in the arms were the first symptom, the fourth month the involuntary movements began. The incoördination by the end of the year now is so extreme that the man is unable to feed or dress himself.

Malan and Civalleri. LESIONS OF OPTIC THALAMUS. [Policlinico, June, 1921, 28, No. 6.]

The only pathologic findings were in the optic thalamus in the case described, in which preparalytic hemichorea had developed suddenly in a coachman of fifty-two, addicted to alcohol.

Muskens, L. J. J. CLINICAL OBSERVATIONS ON FORCED MOVEMENTS IN THE HORIZONTAL PLANE THROUGH LESION OF THE CENTRAL VESTIBULAR CONNECTIONS IN THE FORE-BRAIN. [Psychiat. en Neurolog. Bladen, 1921, No. 3 and 4, May-August, p. 248.]

Muskens has previously shown that in a lesion of one-half of the pons up to the anterior corpora quadrigemina the manège movement is from the damaged side towards the sound, but that it tends towards the damaged side as soon as the lesion is made orally of the posterior commissure. In a wound of the thalamus, lamina externa thalami or the globus pallidus or putamen striati one sees in quadrupeds manège movement towards the affected side, but this is much less durable and less violent than when the lesion is in the dorsal part of the brain stem (*i.e.*, the posterior longitudinal bundle). Only when the lesion involved the thalamus and the posterior longitudinal bundle of one side (say R.) was the latter predominant so that the manège movement was towards the left. Muskens records several clinical cases, mostly verified by necropsy, from which it is seen how regularly, in cases showing manège movement or conjugate lateral deviation, the lesion is situated in the basal ganglia or the brain stem; further, one sees how the symptoms of manège movement, head movement, and conjugate deviation of the eyes to one side are connected together, indeed they often occur together, though they can also be isolated. This was long ago shown by Broadbent in dogs. The paths for this series of forced positions and movements are not identical, though they may well lie close together. On the basis of the anatomo-physiological work on forced movements we can reduce all three deviations to a lesion of the central connections of the vestibular nerve. In those patients who showed conjugate deviation of head and eyes a lesion was invariably found in the lenticular nucleus or the globus pallidus or their connections with the commissural region (stria-commissural connections). One of Muskens' cases illustrates the fact that forced movement in the horizontal plane (manège movement with forced position of head and eyes to the side) may be of less value in the determination of the side of the lesion than is the direction of falling to the side. [LEONARD J. KIDD, London, England.]

6. ENCEPHALITIS

Lépine. THE SOIL IN INFECTIOUS ENCEPHALITIS. [Bull. de l'Acad. de Méd., October, 1920, 84, No. 32.]

In this thoughtful paper Lépine advances an hypothesis as to the reasons why epidemic encephalitis is not more actively contagious. In fifty cases analyzed he emphasizes environmental factors. These were either excessive physical strain or emotional unrest, a constitutional nervous or mental background or the onset or conclusion of puberty or the menopause. In a discussion that followed Netter stated that he believed a number of abortive and light cases were always to be found

but it was only when the nervous system is substandard does the disease manifest itself as encephalitis. Pregnancy seems to offer peculiar stresses favorable to encephalic lesions.

Bab, Werner. PSYCHOGENIC COMPONENTS IN THE ORIGIN OF DOUBLE IMAGES IN A CASE OF ENCEPHALITIS LETHARGICA. [Neurologisches Centralblatt, 1920, No. 12.]

Report of a case in which the lethargic state had a peculiar effect upon the eye muscle disturbance frequently observed in encephalitis lethargica. The thirty-year-old patient was under treatment from March 4 to April 12, 1920. A vigorous chill had preceded and on March 3 double vision had set in. The eyes showed beside a slight myopia (while acuteness of vision was good) a slight flattening and inequality of the narrow pupils. The pupillary reactions were good. On both sides slight ptosis. No anomalies of position, no defect of mobility of the eye muscles. At the lateral end positions brief, flashing, jerking nystagmus. Refractive media clear, no pathologic findings by the ophthalmoscope. In a brief preliminary test for double images in which a red disk was placed before the right eye there were double images in the sense of a paralysis of the musculus rectus lateralis dexter.

The psychic condition was typical for encephalitis lethargica, lowered motility, dull, sleepy facial expression, diminished speech reactions, difficulty in comprehension, impaired attention, stumbling over syllables as in disturbances of articulation. Nothing especial could be determined neurologically. On making a further test for double images in a darkened room the patient spontaneously explained that he saw the finger double which was held before him and that the right image was red, the left white. This time no red disk had been held before him. He adhered to his statement even at the suggestion that a red image can be produced only through a red glass. In spite of urgent questioning we did not succeed on further testing with a red glass before the right eye in establishing a uniform type. The double images changed continuously. The right abducens paralysis discovered at first could no longer be proved. The double images appeared always the same in nature but in all directions, laterally as well as above and below. Their distance changed very much. Finally the exposed light was no longer seen simply anywhere. In a few days the double images receded, the psychic state returned to normal and the patient was well. The diagnosis was made as encephalitis lethargica in spite of the positive result of the Wassermann reaction because of the preceding febrile illness occurring during an epidemic of gripe and immediately followed by the temporary organic-nervous symptom picture. The peculiarity of the case lay in the appearance of colored double images without the employment of a colored glass in the test, also in the constant change in the form of the double images so that no type could be established and finally in the rapid disappearance of the symptom which had existed in all for five days. The diplopia owes its tem-

porary and changing character to a slight, diffuse disease in the nuclear region of the brain. The extraordinary fact was that the psychic condition of the patient complicated the otherwise easily explained symptom of diplopia. The drowsiness, the little active condition, the lack of attention, and the difficulty in comprehension combined to bring about the colored double images without external cause. This peculiar phenomenon is to be understood only in the sense of these psychic components. [Author's abstract.]

Legry and Lermoyez. CARBON MONOXID POISONING. [Presse Médicale, Nov. 13, 1920, 28, No. 83. J. A. M. A.]

The blood in the cerebrospinal fluid may reveal the carbon monoxid long after it has disappeared from the blood stream. In a case described the lumbar puncture fluid was almost pure blood, confirming the assumption of a congestive and hemorrhagic process in cortex and meninges as part of the reaction to the poison.

Hanns. LETHARGIC ENCEPHALITIS. [Prog. Méd., Oct. 30, 1920, 35, No. 44.]

In this clinical paper the author believes he has something new to report. This patient, a young woman, first developed an infectious sore throat with an eruption on the limbs and intense meningeal reaction but no pronounced lymphocytosis in the spinal fluid. In addition to this an intense conjunctivitis with edema and ptosis of the lids and double bronchopneumonia developed. The temperature was slightly above normal and the pulse was normal. There was some restlessness which was followed by lethargy. In two weeks she was well.

Maggiore and Sindoni. ETIOLOGY OF EPIDEMIC ENCEPHALITIS. [Pediatrics, Nov. 1, 1920, 28, No. 21.]

These authors are convinced of the close affinity of this syndromic trend to influenza and to poliomyelitis and have already reported a certain amount of success in treating epidemic encephalitis with an anti-influenza bacillus vaccine. They here report on isolating from the blood and spinal fluid of five patients with different types of epidemic encephalitis a gram-negative coccus with which they state they have reproduced the disease in rabbits by intracranial injection of a patient's spinal fluid or intravenous injection of a culture of the coccus. The same clinical picture and pathologic anatomic findings was repeatedly obtained in passing the infection through animals. The cocci resemble the germs isolated by Noguchi in epidemic poliomyelitis. There is, however, a greater susceptibility of rabbits and of adults to this encephalitis coccus. The several clinical types of epidemic poliomyelitis as laid down by Wickmann and others find their precise analogues in epidemic encephalitis, and the pathologic anatomy is closely alike, they say, especially the neurophagia and the polyblastic infiltration of the pia.

BOOK REVIEWS

Mitchell, T. W. MEDICAL PSYCHOLOGY AND PSYCHICAL RESEARCH. [E. P. Dutton and Company, New York.]

A book of this sort does much to establish the term "psychical research" in its rightful place as a field of scientific investigation. Mitchell chooses certain phenomena to represent the problems with which psychical research must occupy itself. They are phenomena not extraordinarily rare in medicine or even in daily affairs. The author assembles his facts concerning them with regard for scientific carefulness in acceptance and statement of fact and with judgment restrained from hasty conclusion. He presents a chapter which studies hypnosis chiefly in setting forth the striking appreciation of time on the part of "somnambules," that is, of persons who after hypnosis are amnesic for all that has happened during the trance. He discusses hysteria and reviews cases of dissociation or "multiple personality." In all he reveals an attitude of open-mindedness toward whatever explanation of facts may prove itself justifiable provided that a full review of facts is submitted to dispassionate observation. The author's own standpoint upon this ground places his book apart as an authoritative presentation of phenomena too often differently set forth. He has not as a rule pressed into the emotional content of the psychic life out of which the manifestations recorded are likely to have arisen. Yet he has not been blind to this but points definitely to such a background for explanation. Much of the material given was no longer capable of investigation upon this side as it is reported from former observers. In one instance Mitchell reports his own use in part of Freud's technic with successful result. His conception of suggestion, of the true nature of hypnosis and of hysteria as well as his discussion of multiple personality shows much agreement with the psychoanalytic point of view. The author inclines to a belief in a unitary soul which can persist behind all the dissociations of consciousness but in this last chapter upon "Body and Soul" he still remains free from dogma, presenting his discussion with an openly inquiring mind.

Grieg, J. Y. T. THE PSYCHOLOGY OF LAUGHTER AND COMEDY. [Dodd, Mead and Company, New York, 1923.]

One may begin reading this book at the Preface or at the Introduction. One will read both and then go on to the end. At either beginning the author takes one cordially by the hand, disarming hostility but not barring criticism or disagreement, and at once thoroughly awakening interest. He has thought out his way clearly so that he states his conclusions with sufficient positiveness but never

with dogmatism. One feels that he is but making inquiry to discover enlightenment for himself and his readers upon a far-reaching group of psychological phenomena but also to keep free thought stimulated upon these matters of laughter and comedy within human society.

Grieg as a psychologist occupies his own independent ground and is able to define psychological data and psychological processes with clearness of detail. Here, too, he is not dogmatic but rather broad in his acknowledgment of the work of others and their influence upon his thought. He nowhere attempts to force the adherence of the reader to his point of view. On such a background he builds up his theories of laughter and comedy. He is largely guided by Freud but not to the exclusion of the influence of other writers nor of his own independent methods of arriving at interpretations and explanations. He rightfully reminds us that Freud chiefly studied wit and left the field open for a broader survey of other forms of laughter phenomena.

Grieg traces the beginning of laughter back to infancy, giving large place to this important earlier phase of laughter. Here he finds it as the embellishment of the love instinct, which in the infant is subject to an uncertain and ill-coördinated behavior. When an obstruction interrupts behavior and then is suddenly overcome, the result is laughter. Laughter can easily pass from being a love manifestation to functioning with hate since love and hate lie in such close relationship. Such is the simple formula of laughter which Grieg tests through the many forms in which it appears in the varied complexities of human life. And always he finds the formula capable of explaining the most complex forms of laughter-provoking phenomena.

His book contains a well arranged bibliography and a valuable review also of theories of laughter and comedy propounded throughout literature ancient and modern.

Pfister, Oskar. SOME APPLICATIONS OF PSYCHO-ANALYSIS. Authorized English Version. [Dodd, Mead and Company, New York; George Allen and Unwin, Ltd., London; 1923.]

This book of Pfister appearing as an extended form of already published matter and now made accessible to readers of English presents much that is savory and wholesome as food for thought. Pfister is a cultured writer. He is a writer who himself delights in thinking and he proves himself as indefatigable in the sphere of comparative study of other men's intellectual work as he is in the practical application of psychoanalysis to all branches of human endeavor. One's own capacity for thinking as well as one's own practical interest in testing psychoanalysis must be stimulated by a careful perusal of his book. It forms an interesting field of information also as the author makes striking comparison between psychoanalysis and the psychology which has confined itself only to consciousness and as he discusses the philosophy of various exponents of psychoanalysis. He shows that Freud is not among these philosophers

since he confines himself to the more strictly empirical phase of psychoanalysis, leaving the field to be worked by others in its further implications. Pfister supports Freud with a loyalty which constantly reaffirms the latter's basic principles as the sure foundation upon which the truth and practicality of further work may be tested. Pfister thinks deeply yet he leaves one with the feeling that he has frequently been more ingenious than clear in his effort to convince himself that one can retain so much as he does of the content and form of accepted religion and yet accept the uncompromising revelations of Freud's investigations into human psychic sources. Pfister inspires profound respect for his breadth of thought no less than for his distinguished success as a psychoanalytic pastor, testimony to which appears also in this volume. Yet doubt whether his personal and professional bias is not forcing psychoanalysis to an undue support of religion mingles with astonishment as one comes for instance to his discussion of psychoanalysis and mission work. Analysis, he states, causes the "dissolution of every other form of religion, whereas the principles of psychoanalysis are perfectly at one with those of Christianity." Pfister it is true is as broad in his Christianity as in his psychoanalysis but his vision is still blurred to some of the facts of Christianity psychoanalytically discerned.

Peterson, F., Haines, W. S., Webster, R. W. et al. **LEGAL MEDICINE AND TOXICOLOGY.** Second Edition. 2 Volumes. [W. B. Saunders Company, Philadelphia and London.]

The first edition of this work was published twenty years ago. It was then and is now the best work of its kind in its particular field. It has been entirely revised, most thoroughly rewritten and brought up to date in an exemplary manner. Much new matter has been introduced so that practically every problem connected with the medico-legal frame of things receives illumined and thorough discussion. There are 33 articles, all more or less closely correlated, written by nearly as many well-recognized students of the matters dealt with. They can not all be particularized in this notice. The range of interests covered may, however, be indicated. They are: Legal Rights and Obligations of Physicians; Identification of the Living; Identity; The Signs of Death; Sudden Death; Death from Cold, Heat and Starvation; Death and Injuries by Lightning and Electricity; Wounds; Gunshot Wounds, Burns and Scalds; Railway Injuries; Injuries and Disorders of the Nervous System Following Railway and Allied Accidents; Speech Disorders; Inebriety; The Stigmata of Degeneration; Mental Disorders in Medicolegal Relations; Mental Defect Group; Mental Perversions of the Sexual Instinct; Malingering and the Feigned Disorders; Summaries of State Laws Relating to the Insane; The Legal Aspects of Pregnancy; Legitimacy—The Determination of Sex—Signs of Delivery; Birth and Legitimacy; Abortion; Infanticide; Impotence and Sterility; Rape; Unnatural Sexual Offences; Marriage and Divorce; The Medicolegal Aspects of Vision and Audition; The Medicolegal Rela-

tions of Venereal and Genito-Urinary Disorders; The Medical Jurisprudence of Life Insurance; The Medical Jurisprudence of Accident Insurance; General Principles of Toxicology; Forensic Questions Relative to Poisoning; The Technic of Medicolegal Postmortem Examinations; Inorganic Poisons; Gaseous Poisons; Death from Asphyxia; Alkaloidal Poisons; Non-Alkaloidal Organic Poisons; Industrial Toxicology; Food Poisoning and Food-Borne Infections; Poisonous Mushrooms; Poisonous Proteins; Postmortem Imhibition of Poisons; The Destruction and Attempted Destruction of the Human Body by Fire and Chemicals; Death from Pounded Glass and Other Mechanical Irritants; Medicolegal Examination of Blood and Blood-Stains; Medicolegal Examination of Seminal Stains; Medicolegal Examination of Hairs; Medicolegal Relations of the x-Rays, Radium and Ultra-violet Rays; The Common Law and Statutory Obligations of Pharmacists.

It is a library in itself and one of sterling value.

Petrén, K. DIABETES STUDIER. [Glydendalske Boghandel, Copenhagen, 1923.]

Petrén's contributions to neurology are well known to our readers. His interesting study of a case of osteoarthropathy following upon an encephalitis lethargica in the JOURNAL indicate Petrén's chief interests, *i.e.*, neurology and metabolism, and their integration. We have had occasion to comment most favorably upon a large and important neurological treatise published a few years ago, and now this present work on metabolism and on diabetes is before us. It contains, given in a somewhat discursive style, his researches on diabetes during the past twenty years. As a pupil of Naunyn's the stimulus of his master shows throughout and the thoroughness of his work is apparent everywhere. Although for the neurologist the question of diabetes may seem of secondary interest, it is evident that the increasing importance of vegetative neurology is causing nearly all metabolic disturbances to impinge upon the neuropsychiatric fields. Petrén is not unmindful of this relationship although the present volume is strictly biochemical in its general trends.

Certain features are emphasized, notably the influence of the nitrogen metabolism in the phenomena of coma. His researches tend to show that dietary regulation, not starvation, can accomplish much in the presence of acidosis and coma; in the treatment a reduction of the nitrogenous intake is imperative; the necessary calories can be obtained from fats with the minimum of carbohydrates. In his general attitudes so far as American research tendencies are concerned he would tend to support Joslyn's more catholic, rather than Allen's more drastic attitudes.

It is a superb book and well merits the attention of all serious students of metabolic disorders.

OBITUARY

DR. EUGÉNE DUPUY

Eugéne Dupuy, M.D., of Paris, former vice-president of the Société de Biologie and member of the Royal Society of London, died February 14, 1924, at his residence in Versailles, France, in his seventy-seventh year.

A native of Mauritius, Dr. Dupuy took his degree in Paris. He had been assistant to Professor Brown-Séquard, both while Brown-Séquard was in the United States and after he succeeded Claude Bernard to the chair of Physiology in Paris; professor of experimental physiology in the Women's Medical School of London. He was an early member of the American Neurological Association and was first vice-president of the New York Neurological Society. He resided at different times in the United States, where he gave a number of lectures on the brain, in Philadelphia, Boston, New York, New Haven, Baltimore, Chicago, etc. His early researches on convulsive states are classical.

Dr. Dupuy will be remembered with great respect as a man of science as well as an enlightened, capable, and conscientious practitioner.

N. B.—All business communications should be made to *Journal of Nervous and Mental Disease*, 64 West 56th St., New York.

All editorial communications should be made to Dr. Smith Ely Jelliffe, Managing Editor, 64 West 56th St., New York.

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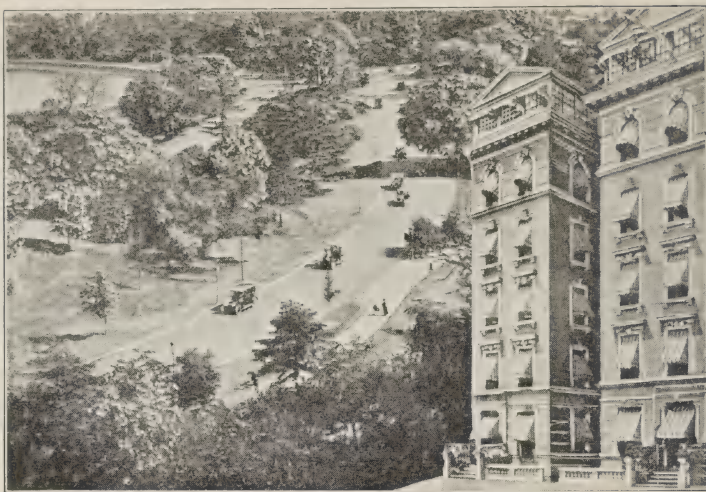
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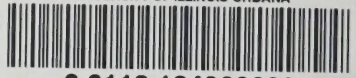
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